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ARCHIVES OF SURGERY

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SUBCUTANEOUS INJURIES OF THE ABDOMINAL VISCERA

ANATOMIC AND CLINICAL CHARACTERISTICS *

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NEW YORK

Two types of lesions are produced by physical violence on the abdominal cavity and its organs. The first is represented by stab wounds and bullet wounds, which penetrate the skin and leave characteristic external marks. They are known as percutaneous injuries. The second type is due to the action of nonpenetrating blunt force, which traumatizes the abdominal viscera but which may or may not leave marks on the surface of the skin. These are called subcutaneous injuries. Of the two, the second variety offers more difficult problems in diagnosis and treatment than the first. While the percutaneous wound can scarcely be overlooked because of the penetration of the skin, the subcutaneous injury often lacks external indications and frequently is not noticed on examination. As many of these injuries are dangerous, though not necessarily hopeless if the patient is given prompt treatment, their early detection is a matter of grave importance to the surgeon.

The subcutaneous abdominal injuries and the symptoms that they present are so varied that it would be impossible to give any set of rules specific enough to serve as a guide for early diagnosis. However, a brief systematic presentation of the different lesions, indicating what can be logically expected from the action of blunt force on the abdomen, might be of value, and such a review is attempted here.

As the type of the abdominal injury depends to a great extent on the nature of the violence, a classification of the different forms of blunt force is necessary. There are two main categories:

(*a*) Generalized violence, or trauma that involves the abdomen as a whole. Examples of this comprise accidents in which the patient is run over by a vehicle, falls from a height, is compressed between two colliding objects or is crushed by a falling mass.

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* From the Medical Examiner's Office of New York City, the First Surgical Division of Bellevue Hospital and the Surgical Service of the Presbyterian Hospital.

(*b*) Localized violence that occurs when an object with a limited area of impact comes in contact with the anterior abdominal wall. Examples of this type include kicks by horses, kicks with the foot, stamping on the body, blows with fist or stick and thrusts with a wagon pole.

The abdominal injuries themselves are separated into the following groups:

1. Injuries of the parenchymatous abdominal viscera, which are represented by the liver, spleen, kidney and pancreas.

2. Injuries of the hollow abdominal viscera, which include the stomach, the intestines and their mesenteries and the urinary bladder.

INJURIES OF THE PARENCHYMATOUS ABDOMINAL VISCERA

The parenchymatous organs lie in the upper part of the abdomen and are more or less protected by their deep position or by the lower ribs and the lumbar spinal column. In the vast majority of cases, they are injured only by a severe grade of violence. Exceptions occur, which will be noted later. The usual results of the injury are marked shock and profuse internal hemorrhage, which is frequently fatal.

A severe generalized trauma sometimes causes multiple ruptures of the abdominal and thoracic viscera, fractures of the ribs, spine and sternum, and other lesions. Reports of a large number of such cases were found among the records obtained at necropsy; as death occurred almost immediately after the accident, it was felt that little was to be gained from any prolonged study of this material. A general statistical survey was not undertaken for this reason, and only selected cases which illustrate characteristically the clinical and anatomic features of the different injuries are presented.

LIVER

Of all the abdominal viscera, the liver is the one most commonly ruptured by nonpenetrating blunt force. Geill[1] estimated that such ruptures occurred in 59.9 per cent of all injuries of the trunk, and others accept his figures as substantially correct.

The anatomic characteristics of the organ explain its susceptibility to trauma. It has a closely knit structure, which is only slightly elastic, and so cannot sustain a marked alteration in form without a break in continuity. It occupies the entire thickness of the right hypochondrium and is partly protected by the lower right ribs. Ligaments attach the liver to the surrounding structures in such a way that its range of motion in any direction is more or less limited. Any force applied to the right side of the upper part of the abdomen, if intense enough, will cause a rupture, as the liver cannot easily escape the effects of the violence. The types of trauma usually described are such severe forms

1. Geill, C.: Vrtljschr. f. gerichtl. Med. **18**:205, 1899.

as occur in accidents in which the patient is run over by a vehicle, falls from a height, is crushed between the bumpers of freight cars or receives thrusts with a wagon pole and kicks (Edler [2]).

The normal organ will be broken only under the application of a marked degree of violence, but in some instances a pathologic condition is present which increases the fragility of the parenchyma. Ruptures may then occur under the influence of such slight trauma as a sudden movement of the trunk; hence, they must be classified as spontaneous. Westenhöffer [3] described such a rupture in a liver that was secondarily involved by carcinoma, and Wätzold [4] mentioned a parenchymatous tear caused by the bursting of an intrahepatic aneurysm. However, these cases are rare.

There is little agreement in the literature concerning the way in which ruptures of the liver are produced. As Walz and Holle [5] have pointed out, the same variety of trauma may produce different injuries in different cases and, conversely, similar lesions may result from dissimilar forms of violence. The exact course of events which takes place at the moment of the casualty must be a matter of conjecture in many instances, and even necropsy may fail to disclose the true modus operandi of the mechanism. This question can be considered more advantageously in connection with the different types of injury.

Hitzrot's [6] classification is perhaps the most suitable. He divides the subcutaneous injuries of the liver into: (1) true ruptures, in which both capsule and parenchyma are torn; (2) subcapsular ruptures in which the lesion is produced below the intact capsule.

The true ruptures of the liver vary widely in size, shape, position and mode of production. The most common varieties are the cracks in the sagittal plane found on the upper convex surface. They occur as shallow fissures, more or less parallel, or as larger single lacerations which divide the parenchyma partially or completely. Some are undoubtedly produced by an extreme bending of the organ in such a way that the diaphragmatic surface is rendered acutely convex and the right and left poles are forced downward toward each other. The result is a break of the liver at right angles to the axis of bending, and consequently the direction of the rupture is in the sagittal plane of the body. Lateral compression of the trunk at the level of the lower ribs causes many lesions of this sort.

According to Walz and Holle,[5] sagittal ruptures on the upper surface follow falls from a height if the body lands on the head or shoulders. If the lungs are in full expiration at the moment of impact,

2. Edler, L.: Arch. f. klin. Chir. **34**:343, 1887.
3. Westenhöffer: München. med. Wchnschr. **51**:41 (Jan. 5) 1904.
4. Wätzold: München. med. Wchnschr. **53**:2107 (Oct. 23) 1906.
5. Walz and Holle: Vrtljschr. f. gerichtl. Med. **40**:215, 1910.
6. Hitzrot, J. M.: Ann. Surg. **66**:50 (July) 1917.

the liver will be driven upward into the funnel-shaped lower part of the chest, which will distort and break it in the manner described.

Under other conditions, falls from a height are supposed to result in the production of sagittal tears of the concave surface. According to Walz and Holle,[5] a fall on the head or shoulders with the lungs in full inspiration will cause the glottis to close spasmodically; it will flatten the diaphragm and keep the liver from entering the chest. The under surface is forced to bulge away from the diaphragm at the moment of impact and thus is ruptured. Falls on the feet or buttocks are also supposed to produce the same result; the diaphragm is flattened on the recoil, and the concave surface bulges downward and sometimes ruptures. During this kind of fall, the ligaments of the liver are not stretched, according to Walz and Holle,[5] and have little or nothing to do in the production of the lesion.

In some instances, a violent trauma which acts tangentially on the epigastrium in an upward direction will force the anterior edge of the left lobe toward the diaphragm. This movement will tear the anterior portion of the concave surface. The point most often involved is the furrow that contains the round ligament.

Sometimes violence applied directly against the front portion of the liver will crush the organ between the anterior abdominal wall and the projecting edge of the vertebral bodies. A complete sagittal rupture at the junction between the right and left lobes is the result. This sort of injury was present in a case of homicide; the abdomen had been stamped on as the woman lay on her back.

Transverse and oblique ruptures are found on both the upper and the under surfaces of the liver. They are due to pressure exerted on the anterior edge in a backward direction. Bending occurs in such a way that the sagittal diameter is shortened and the upper or under surface is rendered acutely convex, which produces the corresponding tears. They are not so common as the sagittal ruptures. The same violence is also supposed to drive the posterior portion of the right lobe against the vertebral column and cause a laceration of the liver in this region. The name of "contrecoup rupture" has been given to this injury, because it occurs at a point opposite the place at which the force is applied.

A common rupture is the craterous laceration found on the upper surface of the right lobe near the coronary ligament. It may be produced by a lateral compression which fractures the right lower ribs and forces them against the parenchyma in such a way as to cause a tear. The sharp ends of the broken ribs may pierce the liver by penetrating the diaphragm, though this complication is rare. The lesion also may be the result of a severe localized violence, such as a kick by a horse, which directly lacerates the organ.

Injuries of the liver in which the capsule is unbroken or subcapsular ruptures are much more rare than the true ruptures. Two varieties are described: (*a*) the subcapsular hematoma and (*b*) the central rupture of the liver.

(*a*) The subcapsular hematoma is a collection of blood between the capsule and the parenchyma. It is usually found on the superior surface of the organ and is probably the result of a force that depresses the liver downward and laterally. The ligaments are put under stress but are not torn. The capsule, however, is stripped from the parenchyma, and blood from the torn vessels tends to accumulate in the newly created space between the two, in larger or smaller amounts. According to Edler,[2] this lesion is accompanied by much local pain, as the capsule is well supplied with nerves.

(*b*) The central ruptures of the liver are closed cavities in the parenchyma filled with blood and traumatized tissue of the liver. Formerly this lesion was thought to be caused by the bursting of a large intrahepatic artery, analogous to the spontaneous hemorrhages that occur in the substance of the brain. In recent years, however, the lesion has been regarded as the outcome of a severe violence that presses the anterior edge of the viscus backward and then moves laterally to the left. The posterior portion is anchored by the spine, and the final result is to twist the liver as one would wring a wash-rag (Bauer).[7] The parenchyma is shattered extensively, with tearing of the vessels and consequent bleeding into the lacerated area. Large intrahepatic hematomas sometimes are formed. The usual site is in the right lobe, but occasionally the left lobe, the interlobar areas or the lobus spigelii may be involved.

The complication that follows rupture of the liver is, first, shock, which probably is referable to the close association of the organ with the sympathetic ganglions. This may be sufficient to cause a temporary period of unconsciousness immediately after the trauma (Orth).[8] Sometimes the patient is prostrated, but in other instances there are few immediate ill effects. In a series of eight cases, three patients were noted as having walked for a short distance immediately after the injury. Auchincloss [9] mentioned a similar case.

The complication that comes next in point of time is that of hemorrhage into the abdominal cavity. This is due to the tearing of the intrahepatic blood vessels in the region of the rupture. In the early stages, the blood is fluid and bright red and may contain a few friable, current-jelly blood clots and small fragmented pieces of liver paren-

7. Bauer, H.: Vrtljschr. f. gerichtl. Med. **56**:33, 1918.
8. Orth, Oscar: Arch. f. klin. Chir. **101**:369, 1913.
9. Auchincloss, H.: Ann. Surg. **80**:626 (Oct.) 1924.

chyma. Bile may also be present in small quantities from the tearing of branches of the bile duct. After a few hours, the peritoneal hemorrhage turns dark red and later brownish red. These changes cause irritation; pigmented brown fibrin is deposited on the serous surface and a low grade peritonitis results. If bacteria gain entrance in some way, either from an injury to a hollow abdominal viscus or through a laparotomy wound, a real infective and suppurative peritonitis will develop.

Occasionally some of the hemorrhage from the rupture may enter the extraperitoneal tissues in larger or smaller amounts, especially if the lesion opens into these structures. Accordingly, retroperitoneal hematomas of varying size are sometimes found near the diaphragm and the lumbar fossa. One such case occurred in the material obtained at necropsy.

The most rapidly fatal hemorrhages are generally associated with complete sagittal ruptures of the liver. Of twelve injuries of the liver that caused death either immediately or within five hours, nine were of this type. In rare instances, however, lesions even of this sort may undergo spontaneous healing (Chiari).[10]

The huge craterous lacerations near the coronary ligament may also bleed rapidly, especially if some large intrahepatic vessel is torn. On the other hand, the less extensive lesions show slower bleeding, and blood clots tend to adhere to the laceration. If the patient lives, thromboses are formed in the torn blood vessels and bile ducts, organization tends to occur, and healing by scar tissue is the result. If the laceration is deep, the edges are deprived of their requisite blood supply and within about twenty-four hours become yellowish white and necrotic, forming the so-called traumatic infarct[11] (fig. 1). Under ordinary conditions these areas are absorbed, but a large piece may become separated from the liver tissue and act as a sequestrum. Occasionally it is enclosed in a cavity formed by adhesions of the rupture to the surrounding structures, and later undergoes transformation into a true abscess (Fertig[12] and Biernath).[13] The process is slow, and takes months for its completion.

The central ruptures of the liver do not bleed into the abdominal cavity and, unless death occurs from the primary shock, complications develop slowly. In some instances the hemorrhage in the substance of the liver may cause a large swelling in the upper part of the abdomen and enough disturbance to justify operative intervention for this reason alone.[6] Small ruptures tend to heal with the formation of central scars; others, a triflle larger, develop traumatic infarcts and

10. Chiari, H.: Berl. klin. Wchnschr. **45**:1629 (Sept. 7) 1908.
11. Orth, J.: Deutsche path. Gesellsch. Verhandl. **3**:82, 1901.
12. Fertig, J.: Deutsche Ztschr. f. Chir. **87**:87, 1907.
13. Biernath, P.: Arch. f. klin. Chir. **90**:73, 1909.

sequestrums. In instances in which the extravasation of blood or bile into the rupture is appreciable, a reaction is caused in the surrounding parenchyma, and a fibrous walled cyst is developed. This, in turn, may become an abscess cavity if bacteria gain an entrance.[7]

An interesting complication of ruptures of the liver is the production of emboli of various sorts, which gain entrance to the hepatic veins and are carried to the right side of the heart and the lungs. Engel [14] mentioned a case of fat embolism caused by a ruptured fatty liver. Others authors cited by Bauer [7] record the transportation of cells from the liver to the lungs in a similar manner.

The appendages of the liver, such as the blood vessels and the different parts of the biliary system, are occasionally ruptured. At

Fig. 1.—Sagittal section of liver showing traumatic infarcts around the edges of a craterous laceration.

necropsy the inferior vena cava sometimes shows partial or complete transverse tears at the level where it emerges from the diaphragm. This is the result of a tremendous force which pushes the liver down and away from the diaphragm, and thus puts traction on the vessel. A rapid, fatal hemorrhage into the abdominal cavity is the usual outcome.

The portal vein and hepatic artery are ruptured rarely, even in cases in which a complete tear of the common bile duct is present. The vessels are elastic, are less firmly anchored to the surrounding structures than the duct and are less liable to suffer from the same violence.

14. Engel, H.: München. med. Wchnschr. **48**:1046 (June 25) 1901.

Thrombosis of the portal vein has been described as the result of an injury. The trauma in some way injures the wall of the vessels, either by a dislocation of the contiguous organs or by direct contusion of the vessel against the spinal column. The thrombosis in time occludes the vein and later, by organization, turns the vessel into a solid fibrous cord. The usual complications are esophageal varices, ascites and similar conditions, all of which are referable to the portal obstruction (Thorel).[15]

Ruptures of the hepatic artery are rare and are found only with the more severe grades of trauma. Just[16] recorded a case of multiple injuries in the upper part of the abdomen in which the cause of death was a delayed hemorrhage from an accessory branch of the hepatic artery which was thought to have been eroded by gastric secretion from a nearby injury to the wall of the stomach.

Ruptures of the biliary system occasionally occur. Any portion of the tract may suffer from the violence. In his analysis of sixty cases from the literature, Lewerenz[17] catalogued the ruptures as follows: gallbladder, 23; intrahepatic bile ducts, 8; common bile duct, 9; hepatic ducts outside the liver, 6; cystic duct, 1 and location of lesion uncertain, 13.

The mechanism of the injury varies with the part of the tract affected. The gallbladder may at times be traumatized by severe violence, or it may be ruptured by a comparatively slight force which compresses the organ and causes it to burst by a hydraulic action. This is especially prone to occur if the wall is diseased or if a marked grade of cholelithiasis is present. Ruptures of the liver are only occasionally associated with this injury.

Three methods of rupture have been made responsible for the lesions of the common bile duct and the extrahepatic branches of the hepatic ducts.

A hydraulic bursting mechanism is said to operate in a few cases. Some of the ducts are probably lacerated by being crushed against the vertebral column. Most of them, however, are undoubtedly the result of a tearing force; the anterior edge of the liver is forcibly elevated toward the diaphragm, and the ducts are, as a consequence, put under tension. A complete or partial break is prone to occur near the pancreas, especially as the common duct is firmly attached to that organ and has a wall of limited elasticity. These ruptures are found mostly in children and young people who have an elastic and easily

15. Thorel: Ergebn. d. allg. Path. **18**:123, 1915.
16. Just, E.: Arch. f. klin. Chir. **140**:518, 1926.
17. Lewerenz: Arch. f. klin. Chir. **71**:111, 1903.

compressible chest wall (Stierlin).[18] Associated ruptures of the parenchyma of the liver are not present, as a rule.

Ruptures of the large intrahepatic bile ducts are also found and are necessarily associated with a break in the substance of the liver. Any wide and deep laceration of the parenchyma will tear some of the larger branches, but the more characteristic lesions are seen in connection with small sagittal ruptures on the under surface of the organ. They begin at the anterior edge of the liver in the furrow in which the round ligament lies and run backward into the hilum, tearing the left hepatic duct across. This force probably elevates the left lobe of the liver toward the right, so that the bile duct is put under stress.

If death does not occur within a short time from shock and intra-abdominal hemorrhage in cases of rupture of the biliary tract, the continued excretion of bile finally dominates the clinical picture. The bile may be extravasated into the retroperitoneal tissues, in rare instances, if it cannot gain access to the peritoneal cavity either because the peritoneal layer is unbroken or because the rupture is surrounded by adhesions. Collections of bile have been seen around the region of the liver behind the ascending and descending colon, and even extending into the chest behind the diaphragm and pleura.

The usual course, however, is for the ruptured duct to secrete the bile in the abdominal cavity in quantities varying with its caliber. The smaller ducts produce only a small amount and later are closed by plugs of fibrin and scar tissue; this closure is analogous to that of the smaller blood vessels. The larger ducts, however, pour out bile continuously and are rarely closed by a spontaneous process. The bile in the abdominal cavity gives rise to a chain of symptoms that develop slowly and are characteristic.

This complication can best be illustrated by a case that came to necropsy. A young man, aged 20, fell into an excavation with his automobile. At first he complained only of pain in the right hypochondrium. At the end of four days, he developed a pronounced jaundice, and bile appeared in the urine. Eight days after the injury the bile pigment disappeared from the skin and urine, but at the same time he developed a steadily increasing ascites which caused pressure symptoms. Abdominal paracentesis showed this to be pure bile. A laparotomy was not performed. He died on the fifteenth day following the trauma.

At necropsy, the body was poorly nourished and pale, but not jaundiced. The abdominal cavity was distended with about 8 liters of yellowish-brown, bile-stained fluid. The entire peritoneum including the diaphragm was covered by an adherent, fibrinous, bile-stained membrane, about 5 mm. thick, which caused marked contraction of all the hollow viscera and numerous sticky adhesions between adjacent structures. The large intestine contained a few pasty, clay-colored stools. The viscera and the body fluids outside the peritoneal cavity were not jaundiced.

18. Stierlin, R.: Deutsche Ztschr. f. Chir. **73**:463, 1904.

The source of the peritoneal fluid was a small sagittal laceration of the under surface of the liver, which was in the furrow in which the round ligament usually lay and ran into the hilum, communicating with a transverse tear of the large left intrahepatic bile duct. The gallbladder and the rest of the biliary tract were collapsed and free from bile.

It is evident that the rupture excreted much bile from the start and that, in the first few days after the trauma, most of this was absorbed and jaundice was produced. Later, the bile caused a nonseptic peritonitis from the chemical irritation, with the formation of an enormous fibrinous exudate, which blocked the pathway of absorption from the serous surface. This condition resulted in the ascites in which the abdominal cavity was filled with bile and the disappearance of the icterus elsewhere. The entire process is a slow one and, though not septic, is marked by much toxic action. Unless means are taken to close the ruptured duct, the continued excretion of bile will inevitably prove fatal. Cases described by Lewerenz,[17] Stierlin [18] and Hildebrandt [19] give practically the same picture.

In the material obtained at necropsy at the Medical Examiner's Office, protocols of twenty-one cases were collected in which there was a rupture of the liver not complicated by another injury of a fatal nature. A study of these cases brought out some interesting points. The causes of the casualties are listed in table 1. Except in a few instances the trauma was severe.

The age incidence (table 2) did not show anything significant.

A consideration of the causes of death in connection with the lesion in the liver and the length of time the patient survived the accident established the following facts:

1. Seven patients died at the scene of the accident. Of these, four showed a complete sagittal laceration of the substance of the liver and two a huge craterous tear on the upper surface of the right lobe. The six patients died of profuse intra-abdominal hemorrhage. The seventh showed some superficial lacerations on the upper surface, lost only a small amount of blood, and evidently died of the shock of the injury combined with a state of acute alcoholism.

2. Eight patients survived the injury from one-half hour to six hours. Five of these sustained complete sagittal ruptures and died of severe intra-abdominal hemorrhage. One craterous laceration of the right lobe and two superficial lacerations of the surface of the liver were found. These patients died of a combination of shock and acute alcoholism.

3. Two patients survived from twelve to twenty-four hours. Both sustained moderately large craterous lacerations of the upper lobe and

19. Hildebrandt: Arch. f. klin. Chir. **81**:646, 1906.

died of intra-abdominal hemorrhage. In one a laparotomy was performed and the laceration was packed with gauze, but this did not halt the hemorrhage.

4. Two patients lived from four to five days after the injury. One had a craterous laceration of the right lobe and died of a combined hemorrhage and effusion of bile into the abdominal cavity. A large traumatic infarct was present about the laceration (fig. 1). The other patient sustained a few superficial lacerations of the right lobe, but bled only a little intra-abdominally. The cause of death was terminal pneumonia, to which a state of chronic alcoholism contributed as much as the rupture of the liver.

TABLE 1.—*Causes of Casualties*

Accidents in which the patient was run over by a vehicle	13
Falls from a height	5
Crushed between a barge and the pier	1
Struck by falling steam shovel	1
A foot stamp on the upper part of the abdomen	1

TABLE 2.—*Age Incidence*

1 to 10 years	6
20 to 30 years	6
31 to 40 years	3
41 to 50 years	2
51 to 60 years	3
61 to 70 years	1

5. Two patients lived eleven days and fifteen days, respectively, after the injury. Both showed superficial sagittal lacerations of the under surface, which involved the main branch of the left intrahepatic bile duct. Death was due to a continuous effusion of bile into the abdomen, in the manner that has already been described. Abdominal section and packing of the lacerated area was tried in one of these patients, but apparently the operation was performed too late to be of benefit.

Adequate conclusions concerning the mortality of ruptures of the liver in general cannot be drawn from these cases. However, it is obvious that a large proportion (33.3 per cent) of the patients died immediately after the accident, and 38.1 per cent died during the first six hours of their admission to the hospital. They were in such a state of collapse that operative measures could not be attempted. Only 28.6 per cent lived a sufficient length of time for an adequate clinical observation to be made, and in only two of these was a laparotomy

tried. Unfortunately, the clinical histories obtainable were too brief to indicate the reasons for the surgeon's judgment.

Nine clinical records which showed the results of the laparotomy were collected. They are as follows:

CLINICAL HISTORIES

CASE 1.—A girl, aged 18, was run over by an automobile. She was delirious and in a state of shock but not comatose when she was admitted to the hospital. A laparotomy was performed shortly after admission. A sagittal laceration of the midportion of the liver was found; this was packed with gauze. She died three hours later, the cause of death being abdominal hemorrhage.

CASE 2.—A boy, aged 13, was run over by an automobile. He was admitted to the hospital in shock and was pale, with a rigid abdomen, especially in the region of the liver. Some of the lower ribs on the right side were fractured. A laparotomy was performed a few hours later. A large, irregular laceration on the superior surface of the liver was found and was packed with gauze; the abdominal cavity was full of blood. Death occurred seven hours after the injury from abdominal hemorrhage. At necropsy, a ruptured spleen was discovered.

CASE 3.—A man, aged 26, fell from a wagon. Immediately after the fall he got up, walked a block and then fainted. When he was admitted he was alcoholic, pale and in shock, with a rigid and tender abdomen, especially in the upper right portion. He also complained of pain in the right shoulder. A laparotomy was performed eight hours after the injury and a craterous rupture of the right lobe was found, which was packed with gauze. He died thirteen hours after the injury from abdominal hemorrhage.

CASE 4.—A man, aged 24, was struck by a board thrown from a saw; he was knocked down but was not unconscious. On admission, he was in shock and was cold, anemic and vomiting. Pain was noticed in the region of the liver and an abrasion was present over the right side of the chest. A laparotomy was performed eight hours after the injury. A sagittal tear between the liver lobes was found, which was packed with gauze. The patient died twenty-four hours after the injury from internal hemorrhage.

CASE 5.—A boy, aged 13, was struck by an automobile. He was not unconscious after the accident, but, on admission, was dyspneic, cyanotic and in a state of shock. Fractures of the sixth to ninth ribs on the right side were found. A laparotomy was performed a few hours after the accident. The liver was torn from the diaphragm, leaving a lacerated surface of the upper portion of the right lobe. This was packed with gauze. The patient died two and one-half days later in sudden collapse. He had a slow abdominal hemorrhage from the injury.

CASE 6.—A girl, aged 13, fell on a rail fence. She vomited and walked about four city blocks to her residence. Her condition for four days subsequent to the injury was not alarming. She began to complain of constant pain over the region of the liver, and her mother noticed that she was becoming somewhat icteric. She was admitted to the hospital four days later, and an abdominal operation was performed the same day. An area of liver was found attached to the diaphragm, separated from the rest of the organ. This was packed with gauze. The blood in the abdominal cavity was evacuated, and the patient recovered in forty-five days. A sinus in the operative scar per-

sisted for a few weeks, but finally healed. One year after the injury, a second laparotomy was performed to loosen fibrous adhesions around the site of the injury. The scar was firm. With the exception of a neurotic condition, the result was good.

CASE 7 (Auchincloss).[8]—A girl, aged 12, was coasting on a sled and collided with a tree. After the injury, she was not unconscious and walked one-half mile to her residence. She was admitted to the hospital six hours later, complaining of pain and tenderness in the upper part of the abdomen and pain in the right shoulder. The skin over the upper right abdominal quadrant was reddened. Fifteen hours after the trauma, an operation was performed, and a horizontal tear of the upper surface of the right lobe was found. About 400 cc. of blood was found in the abdomen. The laceration was packed with gauze. The patient recovered in thirty-one days and, aside from slight bulging of the scar, was in a good condition.

CASE 8.—A man, aged 48, who was struck by an automobile, was unconscious after the injury, but walked to the ambulance. On admission to the hospital, he showed abdominal pain and rigidity and pain in the right shoulder. A preliminary abdominal tap showed the presence of blood in the cavity. A laparotomy was performed twelve hours after the trauma. A vertical linear tear of the right lobe of the liver was found, which was packed with gauze. The blood was evacuated, and the patient recovered twenty-two days after the accident.

CASE 9.—A girl, aged 9 years, fell 12 feet onto a stone floor. She entered the hospital in shock and was pale and restless, showing a rapid pulse and complaining of pain in the abdomen, especially on respiration. Tenderness and rigidity of the right upper abdominal quadrant were present. A few hours later, a laparotomy was performed. Blood was present in the abdomen and a rupture of the right lobe was found, which was packed with gauze. She recovered about twenty days after the trauma.

These cases illustrate several of the different clinical signs and symptoms that characterize injuries of the liver. Different degrees of shock were noted. In a few instances the patient was knocked unconscious and was in severe collapse. In others the immediate effects of the injury were not alarming, four of the patients being able to walk a greater or less distance.

Signs of abdominal hemorrhage were frequently present, as indicated by pallor, restlessness, cold skin and dyspnea.

Rigidity and pain in the abdomen, especially in the region of the liver, were noted in all of the cases except those in which marked shock was present. Pain referred to the right shoulder was present in three cases. This symptom has often been emphasized in the literature as typical of an injury to the liver.

External indications of injury to the region of the liver were evident in four instances. Two patients had fractures of the ribs on the right side near the right hypochondrium. One had an abrasion of the lower right quadrant of the chest anteriorly; a fourth showed an erythematous area at this point.

In all of the cases the indication for operation was based on the general clinical picture rather than reliance on any special signs. The diagnosis of intra-abdominal hemorrhage was correctly made, and the injury of the liver was suspected, as a rule. In one instance the presence of blood in the abdomen was determined before operation by paracentesis.

The object of the operation was to stop the bleeding from the wound in the liver, and this was accomplished by laparotomy and gauze tamponade of the injured area. In the patients who recovered, this measure seemed to answer the purpose sufficiently well.

From the review of these cases, an idea can be formed of the general characteristics of subcutaneous ruptures of the liver. A large proportion of the patients undoubtedly died soon after the accident from shock and intra-abdominal hemorrhage. The lacerations are either marked or else the patient is suffering from acute alcoholism at the moment of trauma and so succumbs readily to shock.

Other ruptures, on the contrary, are slight and may heal completely under merely expectant treatment. However, such ruptures give few signs and probably pass unrecognized during life. For this reason it is hard to compare them with the other more severe forms.

A third type of patient, however, may live for varying periods of several hours to days, and finally may succumb to the continued flow of blood or bile into the abdominal cavity. Some of them might possibly recover if a diagnosis could be made and the appropriate operative procedures instituted, but others probably could not be saved in any event. The mortality is high, even with operation. Finsterer [20] gave the mortality as about 60 or 80 per cent from the estimates of other authors.

In any event, a prompt diagnosis is a matter of great importance and may be difficult in an individual case. External signs of violence are apt to be lacking. The abdominal symptoms are often characteristic, but occasionally may be obscured by other factors, such as acute alcoholism. General rules cannot be formulated, and the recognition of the injury depends on the acumen of the surgeon.

SPLEEN

Among the subcutaneous abdominal ruptures, those of the spleen hold the second place numerically. Geill [1] found that they occurred in 33 per cent of all cases of visceral injury. The physical characters of the organ and its protected position in the abdominal cavity explain to a certain extent why it is not as frequently involved as the liver by violence applied to the upper part of the abdomen. The normal spleen

20. Finsterer, H.: Deutsche Ztschr. f. Chir. **118**:1, 1912.

is a small vascular organ, varying in weight from 50 to 350 Gm. It is covered by the left vault of the diaphragm and guarded by the tenth and eleventh ribs on the left side. Its long axis is parallel to the curve of these ribs, and its upper pole is situated about 2 or 3 cm. distant from the body of the eleventh dorsal vertebra. Although it is attached by its ligaments to the surrounding structures, it enjoys a certain degree of mobility. All of these conditions work in such a manner that the spleen escapes the results of the violence in many instances.

The normal spleen, as a rule, is reached only by a relatively severe trauma. The mechanism of the rupture has been explained in various ways. Some believe that, with a sudden localized force, such as a blow or a kick acting on the left hypochondrium, the spleen bursts by hydraulic action, especially when its blood vessels are engorged and its pulp is friable. With the sudden increase of internal pressure imparted by the impact, the capsule, which is already tense, will be overstretched and will rupture at its weakest point. Berger [21] declared that this injury is exactly analogous to ruptures of a hollow viscus, such as a urinary bladder fully distended by urine.

Other forms of violence cause the spleen either to bend in the direction of its longitudinal axis, in which case the rupture will be across the organ, or to bend in the transverse axis so that longitudinal ruptures will be produced. The ruptures invariably occur at right angles to the axis of bending on the surface which is rendered convex.

The most common lesions are transverse ruptures of the surface of the hilum, which means that the spleen is overextended or so bent that the surface of the hilum is rendered acutely convex in the longitudinal axis. Several ways have been described in which this result can be attained. In young persons with easily compressible chest walls, a sudden force applied to the left side will drive the ribs against the convex surface of the spleen and cause the surface of the hilum to bow to the right. The action seems to be comparable to the sudden protrusion of the abdomen which follows a violent shove in the small of the back. In other instances a severe tangential force, which travels across the epigastrium from right to left, may drag the lower pole of the spleen below and beyond the lower margin of the left ribs, thus producing the overextension.

Transverse ruptures of the convex surface are caused by overflexion of the spleen. This is supposed to occur as a result of a severe violence which compresses the left side of the chest and catches the two poles of the spleen between the ribs and the body of the eleventh dorsal vertebra.

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21. Berger, E.: Arch. f. klin. Chir. **68**:768, 1902.

A moderate grade of trauma will produce the bending and the simple ruptures that have been previously described. A marked grade of trauma, however, may crush the spleen into many pieces and at the same time fracture many of the left lower ribs.

The different splenic attachments are supposed to have some influence in the production of many splenic ruptures, especially those which accompany falls from a height. The normal ligaments are peritoneal reflections from the contiguous structures, such as the stomach, transverse colon and diaphragm. Occasional aberrent attachments also occur. At the moment of impact, the spleen has a violent impulse to move, but is checked by the ligaments, so that a parenchymatous tear results near the ligamentary attachments.

The spleen is often lacerated by the broken ends of fractured ribs which penetrate the diaphragm, or it may be torn or in other ways crushed by fractured ribs which do not penetrate. In other instances, a direct localized violence may tear the organ (St. John).[22]

Brogsitter [23] described these mechanisms in some detail, emphasizing, however, that in any case it might be difficult to explain the exact way in which the injury was produced.

Spontaneus ruptures of the spleen or ruptures produced by a slight degree of violence are relatively common. In many normal and diseased conditions the organ is large, swollen and engorged with blood, and shows a marked increase in its cellular elements. At such times, the parenchyma is friable and the spleen is especially vulnerable to every slight trauma. Ruptures have occurred during coughing, laughing, sneezing, lifting of weights and similar acts.

The conditions that predispose to spontaneous rupture are predominantly the splenic enlargements in malaria and, to a less extent, in typhoid fever, leukemia, typhus fever, cirrhosis of the liver, the last month of pregnancy and similar conditions (Vorwerk).[24]

Anatomically, the ruptures of the spleen are classified as: (1) ruptures involving both capsule and parenchyma and (2) ruptures involving the parenchyma alone without a capsular tear. Berger called them contusions of the spleen.

Ruptures involving both capsule and parenchyma are the most common. They vary in size from mere jagged splits of the capsule to deep lacerations. Most of them are transverse on the surface of the hilum, though occasionally some are found on the convex surface. A few run longitudinally. Some ruptures are multiple and may be parallel to each other, but more often they take a V, Y, T, H or stellate

22. St. John, F. B.: Ann. Surg. **80**:624 (Oct.) 1924.
23. Brogsitter, C. M.: Charité ann. **33**:494, 1909.
24. Vorwerk: Deutsche Ztschr. f. Chir. **111**:125, 1911.

form. When the force has been particularly intense, the spleen is often found fragmented and its ligaments torn in varying degrees (fig. 2).

The immediate complication following the rupture is hemorrhage into the abdominal cavity. This may be slight or sufficient to cause death. If the bleeding is slow, a blood clot may be attached to the rupture and may surround the organ. During the first few hours the blood retains its normal color, but later undergoes the same changes that were described before. If infecting bacteria gain entrance in any way, a septic peritonitis will result. In some instances in which the spleen becomes adherent to the surrounding structures, a subphrenic abscess may develop.

The small splenic ruptures tend to undergo healing with the formation of scars. Occasionally, anemic traumatic infarcts are encountered

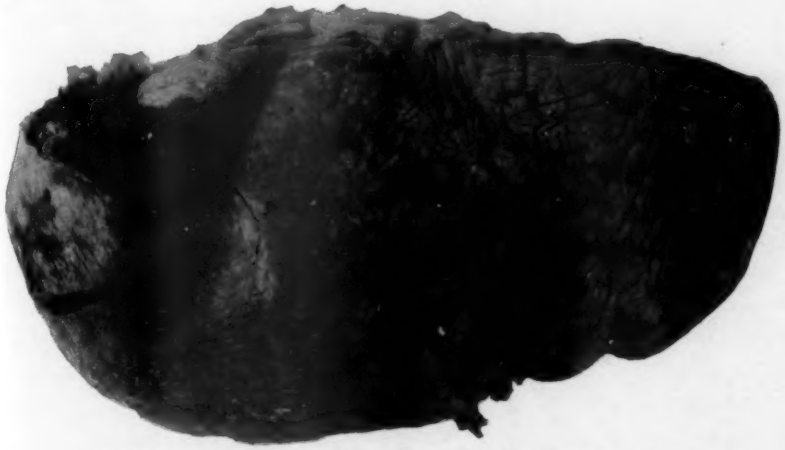

Fig. 2.—Y-shaped rupture on convex surface of spleen.

around the edges of larger lacerations, analogous to the lesions of the liver that have been described.

The contusion of the spleen is the result of a violence that ruptures the parenchyma without breaking the capsule, so that a hematoma is formed in the substance. The effusion of blood may be sufficient to cause an appreciable enlargement of the organ. Blood may be extravasated into the perisplenic tissues. Occasionally, inflammatory changes take place in the capsule and the spleen may become adherent to the surrounding viscera.

The symptoms of the contusion are described as: (1) severe pain in the left hypochondrium; (2) splenic enlargement and (3) a daily fever of the quotidian type.

The pain and tumor are easily explainable on the basis of the anatomic changes. The pyrexia, however, is not so easily accounted

for, and the suggestion that in many instances the injury has stirred up an old septic process or a preexisting malaria is worthy of consideration.

Many sequelae to splenic contusions are described. In the older literature, chronic splenic hypertrophy, either of the simple type or of the leukemic variety, is mentioned as a result of a contusion, but in recent years the tendency has been to doubt this conclusion.

The more usual results are local changes in the splenic parenchyma. Small injuries usually heal by absorption of the blood and the formation of an internal scirrhous scar. Larger effusions develop into true traumatic cysts filled with transformed brownish blood. In rare instances they can become infected by bacteria transported through the arterial blood, and encapsulated abscesses result. If the parenchyma is much damaged, large traumatic infarcts occur, which can serve as a nidus for future infection and may lead to a fatal termination.

A glance at the ruptures of the spleen, as they occurred in the material obtained at necropsy, brought out the fact that the majority were combined with other internal injuries. It was difficult, in a study of these cases, to reach any satisfactory conclusion concerning the effect of the splenic lesion on the system. Only a few isolated ruptures were discovered, and most of them were included among the clinical cases in which operation had been performed. Only three patients had died without operation, and these cases merely served to point out the fact that death was likely to occur after a lapse of several hours from an intra-abdominal hemorrhage. Trauma to this organ rarely produced such rapidly fatal results as were characteristic of some hepatic injuries.

The clinical cases follow:

CLINICAL HISTORIES

CASE 1.—A boy, aged 16, while coasting, struck his left side against a tree. He was not unconscious after the injury and was able to walk, though he felt a trifle dizzy. On admission to the hospital, he complained of pain in the left part of the abdomen, thirst and shortness of breath; he appeared pale. A laparotomy was performed a few hours later. The spleen was found lacerated completely across and was removed. The patient died twenty-one hours after the accident from weakness and loss of blood.

CASE 2.—A man, aged 21, fell 50 feet. He was not unconscious after the injury. On admission to the hospital, a contusion was present over the region of the left flank. He was pale; the abdomen was rigid, especially in the left upper portion, and blood was found in the urine. The left femur also was fractured. An operation was performed and the spleen was found to be ruptured near its lower pole; it was removed. The abdominal hemorrhage was removed. A rupture of the left kidney was found, and the kidney was removed. The wounds later became infected, and death occurred five days after the accident, from streptococcic septicemia.

CASE 3.—A man, aged 26, was crushed in an elevator. He was admitted to the hospital in a state of shock with cramplike pains in the abdomen. The

bones of the left leg were fractured. A laparotomy was performed the same day. The lower pole of the spleen was torn across, and there was bleeding into the splenic pedicle. The spleen was removed. Death occurred fifteen hours after the injury from secondary anemia, due to the primary internal hemorrhage.

CASE 4.—A man, aged 60, while in an alcoholic state, was run over by a wagon. He walked home and some time later collapsed in his bathroom. He was admitted to the hospital twelve hours after the accident, complaining of pain on respiration, anemia and a distended and tender abdomen, especially in the left upper quadrant. Splenectomy was performed eighteen hours after the trauma, and blood was removed. Transverse ruptures on the surface of the hilum were found. Death occurred four days after the accident from lobar pneumonia. A small subcapsular rupture of the left kidney was found, but was not of great significance.

CASE 5.—A boy, aged 13, while coasting down a hill, struck a stone wall. He was able to walk, though he complained of pain in his left side. He was admitted to the hospital thirty-three hours after the accident, complaining of pain in the left side, especially on respiration. Shifting dulness was present in the flanks. A laparotomy was performed one hour after admission and thirty-four hours after the injury. A small rupture of the anterior edge of the spleen was found. The spleen was not removed, as the rupture was not bleeding. About 140 cc. of blood was present in the abdominal cavity, and this was removed. The patient recovered in ten days, without any sequelae.

CASE 6.—A girl, aged 7, fell against the corner of a desk.[22] She walked home and later vomited. She was admitted to the hospital four hours after the accident, suffering from abdominal pain. The upper left abdominal quadrant was rigid. Dulness was present in the flanks. Splenectomy was performed five hours after the accident. An abdominal hemorrhage and a stellate laceration of the spleen were found. Recovery occurred in thirty-five days without any sequelae.

CASE 7.—A boy, aged 13, was run over by a wagon. He entered the hospital in severe shock, but was not unconscious. He was pale and suffering from thirst. The upper part of the abdomen was rigid. The spleen was removed at laparotomy shortly after admission, and was found fragmented. Blood was present in the abdominal cavity. Recovery occurred in thirty-four days, without any sequelae.

CASE 8.—A boy, aged 17, was crushed in the region of the lower part of the chest. He was admitted in a state of shock; he was pale and drowsy and complained of severe pain in the abdomen. Splenectomy was performed shortly after admission. Hemorrhage was present in the abdomen. The spleen was ruptured on the surface of the hilum. The patient recovered in sixteen days.

CASE 9.—A man, aged 23, fell 30 feet down an elevator shaft. He was admitted to the hospital in a semistuporous condition and in a state of shock, with a tender left flank and a rigid abdomen. Luxation of the left shoulder joint was present. Splenectomy was performed shortly after admission, and the luxation was reduced. A transverse laceration of the spleen was found. Blood was present in the abdomen. Recovery occurred in twenty-seven days.

CASE 10.—A man, aged 29, was struck by an automobile one-half hour prior to admission to the hospital. He was admitted in a state of shock with a rigid abdomen and shifting dulness in the left flank. He complained of pain in the left upper abdominal quadrant. Splenectomy was performed shortly

after admission. A deep laceration was present across the lower pole of the spleen. Blood was present in the abdomen. He recovered in eighteen days.

CASE 11.—A man, aged 39, fell 20 feet from a scaffold, landing on his left side. He was pale, showed a rigid and tender abdomen and complained of pain in the left side, especially on respiration. Splenectomy was performed about twenty-four hours after the trauma. Considerable blood was present in the abdominal cavity. He recovered in thirty-two days, the only sequela being a slight hernia in the scar.

CASE 12.—A man, aged 42, was run over by an automobile. He was admitted to the hospital in a state of shock, with severe abdominal pain, especially in the left lumbar region. Luxation was present at the outer end of the right clavicle. Splenectomy was performed the same day, and the blood in the abdominal cavity was removed. A transverse laceration of the spleen was present. The luxation of the right clavicle was reduced. The patient recovered in thirty days, but developed an abscess in the abdominal scar that healed and left a small hernial protrusion.

Of the twelve patients described, four died and eight recovered, which seems to indicate that the mortality is considerably less in ruptures of the spleen than in ruptures of the liver, especially if the patient survives long enough to receive surgical treatment.

These cases illustrated most of the important clinical signs associated with the injury. Shock was sometimes present, but was never overwhelming; four patients were described as being able to walk after the accident. The signs of internal hemorrhage were present, the patients showing pale skin, rapid pulse, dyspnea and thirst. Abdominal pain, rigidity and tenderness were present and often were especially marked near the left hypochondrium. In a few instances, the pain was aggravated by respiration. In three cases, the intraperitoneal hemorrhage gave the sign of shifting dulness in the flanks.

In three cases (4, 5 and 6), the symptoms apparently were not alarming immediately after the accident, and the patients did not enter the hospital until a few hours had elapsed.

The injuries described were of varying grades of severity, from superficial lacerations to fragmentation of the splenic pulp. Splenectomies were performed on eleven patients, four of whom died. One case showed slight laceration of the spleen, which was not bleeding at the time of operation, and so the spleen was not removed; the patient recovered without incident. This case perhaps suggests an explanation for some of the good results that have attended such operative procedures of the past as tamponade of the rupture and suture of the lesion in situ. It merely indicates that a few splenic injuries are not necessarily fatal and would probably recover spontaneously. However, the treatment of choice is splenectomy, as it gives the only adequate assurance that the laceration itself will no longer bleed. Tamponade or suture of a severe injury in such a vascular organ as the

spleen would be futile in most cases. The mortality of splenectomy has been estimated at about 34 or 43 per cent, which agrees with the results in this series.

In the literature,[24] a splenectomy is said to be followed often by some distinctive changes in the blood, the lymph nodes and the thyroid gland. These changes are not constant, however. Swelling of the thyroid has been mentioned, but at present its connection with the removal of the spleen is considered dubious. Pain in the long bones has been occasionally observed. Enlargement of the lymph nodes, especially the supraclavicular, cubital and inguinal groups, has been noted more often, but does not always occur.

The changes in the circulating blood are sometimes pronounced, but at other times are well within the normal variation. The red blood cells may be decreased in number or increased above the usual limits. The hemoglobin may be diminished, and the red cells may appear pale and show the changes of secondary anemia. The leukocyte count may vary from the normal limits to from 40,000 to 60,000. In some instances a marked lymphocytosis, as high as 44.75 per cent, a slight eosinophilia, and a rise in the proportion of blood platelets are present, but these manifestations are not invariably present. All that can be said at present is that the removal of the spleen creates distinct disturbances of a minor nature in the lymphatic hemopoietic system that may be manifested in the manner described. In the series of cases described here, these phenomena were not observed.

In the four fatal cases, death was caused either by the intra-abdominal hemorrhage or by intercurrent infections, which were furthered by the secondary anemia from the hemorrhage. In general, the trauma was severe and other injuries, such as a fractured femur or a ruptured left kidney, contributed appreciably to the fatal result.

KIDNEYS

The kidneys are not so commonly ruptured by blunt force as either the liver or the spleen. Geill [1] mentioned that such injuries comprised 21.5 per cent of all visceral injuries in his collection. As a rule, the lesion is unilateral, and it is rare to find both kidneys involved in the same case.

Their anatomic position in the body explains many peculiarities of the rupture. They are paired organs, lying deep in each lumbar fossa at about the level of the second lumbar vertebra. The blood vessels and ureter enter the hilum, which is placed in the midportion of the internal edge of each. Posteriorly, the right and left twelfth rib cross the middle portion of the corresponding kidney obliquely. Anteriorly, they are covered by a layer of peritoneum, and the greater part of the organ is definitely retroperitoneal.

Structurally, they are vascular parenchymatous masses which, under normal conditions, have a consistency about that of the normal liver. They have the customary reniform shape, with an average weight of about 150 Gm. Each is closely invested by a firm fibrous capsule which is, in turn, surrounded by adipose tissue in greater or less quantity. In the adult, this investment is usually generous, while in the child it is scanty. As the perirenal fat protects the organ from violence to a certain extent, its amount has an important bearing on the complications of the rupture.

The kidneys are ruptured either by a severe trauma or by a relatively slight one. From their anatomic position, it is probable that the less intense grades of violence rupture the organ when applied to the lumbar regions, while only the very marked grades produce their effects by acting on the anterior abdominal wall.

According to Kuester (cited by Boettiger),[25] the basic factor in the production of the lesion is a bursting of the parenchyma due to a sort of hydraulic mechanism. In his experiments on the cadaver, he noticed that kidneys engorged with blood were more easily and more deeply wounded than those comparatively bloodless. It is probable that in many instances a moderate grade of violence will produce a marked lesion if the organ is markedly congested. When the parenchyma is rendered especially friable, because of a pathologic condition like renal tuberculosis or a suppurative inflammation, the rupture may occur under such a slight trauma that it can be classified as spontaneous.[25]

The majority of ruptures are transverse and are directed more or less at right angles to the longitudinal axis. They are probably the result of a force that bends the organ so that the anterior or posterior surface is rendered sharply convex. Several explanations have been advanced to account for this mechanism.

A localized violence, like a blow or kick over the twelfth rib, will drive the rib against the middle of the kidney and either tear the parenchyma directly on the posterior surface or cause the anterior surface to bulge sharply forward and rupture. However, any force that adducts this rib will produce a similar effect, even a sudden movement of the muscles of the trunk.

Some lacerations are said to be the result of a contrecoup violence. A severe force applied to the flank is supposed to crush the kidney against the lumbar vertebrae and extensively lacerate the parenchyma. In any case, it would be difficult to determine just what mechanisms were operative at the time of the trauma, and the question is of theoretical rather than of practical interest.

25. Cited by Boettiger, K.: Deutsche Ztschr. f. d. ges. gerichtl. Med. **8**:33, 1926.

The lesions can best be classified according to Wagner's [26] scheme:

1. Separation of the fatty capsule from the fibrous capsule may occur without rupture of the parenchyma of the kidney. This is caused by a violence which in some way kneads the fat loose, so that a hematoma forms in between the two layers. The blood may be absorbed later and the site of the unjury undergo induration of the connective tissue or, if the hemorrhage is a large one, a perirenal pocket of varying size, similar to a bursa will form,

2. Ruptures of the substance of the kidney which do not involve the pelvis or pyramids are often found. They are usually small transverse lesions on the anterior surface of the organ. If the fibrous capsule is torn, a perirenal hematoma, not accompanied by urinary extravasation, is the result. Small tears generally heal with the forma-

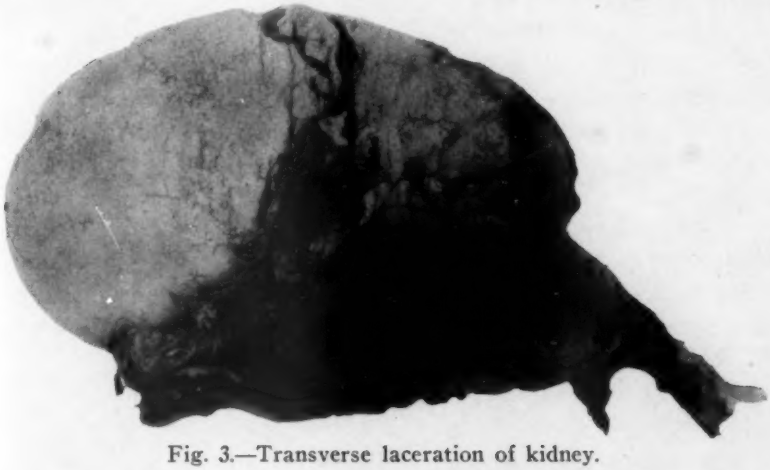

Fig. 3.—Transverse laceration of kidney.

tion of scars. Larger ones may form small traumatic infarcts in the parenchyma. Sometimes the fibrous capsule is uninjured, though the parenchyma may be severely torn. The hematoma may extend to the hilum of the kidney and spread down the spermatic vessels on that side. They have been described as extending to the inguinal ring and pointing into the groin in the course of two or three weeks.

3. Severe ruptures of the kidney, involving either pelvis or pyramids, are occasionally present. They are usually extensive and tend to divide the organ into two parts (fig. 3). A perirenal effusion of blood and urine occurs and may reach a large size. The urine excreted through the bladder may be bloody.

4. Some ruptures are extensive and shatter the kidney into fragments. Generally the same complications follow as were described in

26. Wagner, P.: Schmidt's Jahrbücher **332**:111 (Oct.) 1920.

group 3, but in a few instances prompt formation of thrombi in the torn vessels may stop the bleeding. The fragmented parenchyma, however, becomes speedily necrotic and, if treatment is not instituted, develops into a septic phlegmon or an abscess.

5. Severe renal trauma often is accompanied by the partial or complete tearing of the blood vessels and the ureter. Severe perirenal hemorrhage, extravasation of urine and necrosis of the kidney occur in varying degrees. A massive swelling may develop in the lumbar region from the perirenal accumulation. The fluid causes an inflammatory reaction and may form a cystlike structure around the kidney, which is called a pseudohydronephrosis by some. This complication develops within a week after the trauma, as a rule.

In a few instances a true hydronephrosis, that is, distension of the renal pelvis and parenchyma by urine, may follow the trauma. The usual causes are a fibrous stricture of the ureter following a direct injury of that duct, an extensive perirenal hematoma which compresses the ureter directly, or some obstruction inside the duct itself, such as a blood clot or a preexisting calculus. Boettiger [25] believed that the blood clots are not potent causes of hydronephrosis, as they tend to disintegrate in a week or so and pass into the bladder. Some hydronephroses are described as having been caused by a traumatic displacement of the kidney from its bed, which results in subsequent kinking and blocking of the ureter. The true hydronephrosis takes weeks or months to form and can thus be distinguished from the pseudohydronephrosis which develops more rapidly.

In rare instances, injuries around the hilum cause traumatic aneurysms of the renal artery. They may reach the size of a child's head and form a large tumor in the flank, which may pulsate and give a systolic murmur. They occur in two forms, one of which originates in the extrarenal portion of the artery, while the other may develop inside the substance of the kidney. Both types cause a more or less pressure atrophy of the kidney.

Only a small proportion of renal injuries result in a severe intra-abdominal hemorrhage—only 5.7 per cent, according to Kuester.[27] These hemorrhages usually occur in children at the age when the perirenal fatty capsule is poorly developed and the kidney is somewhat nearer the anterior abdominal wall than it is later in life. The violence is necessarily severe and tears the peritoneal layer over the kidney as well as the kidney itself. Death usually results from the intra-abdominal hemorrhage, but in a few instances, when the renal pelvis is torn, urine will be extravasated with the blood and will cause a

27. Cited by DeQuervain, F.: Deutsche Ztschr. f. Chir. **62**:59, 1902.

peritonitis. The mortality is high, though some of the less severe ruptures of this type are not necessarily fatal.

The subcutaneous renal injuries described in the records of the necropsies were not especially satisfactory for the purposes of study. Most of them were associated with other visceral ruptures and had only a minor influence on the course of events. In only two cases, which occurred in children, were the ruptures of the kidney of any special significance. In one, the peritoneal reflection was torn and the kidney lacerated in a stellate fashion on its anterior surface, causing a fatal intra-abdominal hemorrhage. In the other case, the child was run over by an automobile and the right kidney was split in many pieces, transversely. A retroperitoneal pocket was formed, which led from a space around the lacerated kidney into the right side of the chest. The result was a fatal hemorrhage into the pleural cavity from the traumatized kidney. Other injuries were not present.

The clinical records follow.

CLINICAL HISTORIES

CASE 1.—A man, aged 33, was injured when the taxicab in which he was riding collided with a pillar of the elevated railroad. He was admitted to the hospital complaining of pain in the right lower quadrant of the chest posteriorly. A contusion over the ninth and tenth ribs on the right side posteriorly and a spasm of the right rectus muscle were present. The urine was bloody. Operation was not performed. He recovered in twelve days.

CASE 2.—A girl, aged 12, scraped her right side while coasting on a sled. She complained of tenderness in the left upper abdominal quadrant; blood was present in the urine. An operation was not performed. She recovered in fifteen days.

CASE 3.—A boy, aged 9, was struck by an automobile. On admission, he complained of pain in the right flank. An operation was performed shortly after the injury, by lumbar incision. The right kidney was lacerated at the junction of the upper and middle thirds on its anterior surface, the rupture extending to the hilum. A similar rupture was present on the posterior surface near the middle of the organ. The peritoneum was opened and a little blood removed. The lacerations were sutured. An uneventful recovery occurred in fourteen days.

CASE 4.—A man, aged 26, fell from a scaffold, striking himself on the right flank. On admission to the hospital, he complained of pain and tenderness in the right side. The urine consisted of almost pure blood. Operation was performed six hours after the trauma. A laparotomy incision in the right rectus muscle disclosed some serosanguineous fluid in the abdomen, a large perirenal hematoma on the right side and an extensively shattered right kidney. A nephrectomy incision was made in the right flank and the kidney removed. The patient recovered within a month without incident, except for a slight pulmonary infection following the operation.

CASE 5.—A man, aged 36, fell five stories. On admission, he had pain in the right lower quadrant of the chest and was in a state of shock. Tenderness to pressure was present over the right flank and the upper part of the

abdomen. The urine was bloody. Operation was performed on the day of admission by a laparotomy incision. Blood was not found in the abdomen. A big perirenal hematoma was present on the right side. The right kidney was ruptured across its midportion. A nephrectomy was performed through the laparotomy opening. The patient recovered in sixteen days.

CASE 6.—A boy, aged 16, while running, fell with his left side against an iron bar. On admission to the hospital two hours later, he complained of pain in the left side. The upper left abdominal quadrant was rigid. A few contusions were present over the eleventh and twelfth ribs on the left side. The urine was bloody. A nephrectomy was performed by lumbar incision. The left kidney was surrounded by a perirenal extravasation of bloody urine. The upper pole of the kidney was nearly severed, the laceration extending to the pelvis. Recovery occurred in sixteen days without important sequelae.

The six clinical cases, in all of which recovery occurred, showed several points of interest.

The symptoms were fairly consistent; pain and tenderness were present in the right or left flanks, as the case might be, and blood was found in the urine in variable quantities. In cases 1 and 2, the diagnosis was made on these points alone and, as the symptoms were not particularly alarming, an operation was not performed. Of course, under these circumstances, the diagnosis of renal injury was scarcely conclusive, as pain in the flanks may result from many causes, and even blood in the urine may be due to many other conditions. The cases are described, however, in order to show that mild forms of injuries to the kidneys are fairly prevalent, and to suggest that not a few of these patients recover under expectant treatment.

These patients who were operated on showed much more severe injuries. One recovered after the moderately severe lacerations of the parenchyma had been sutured. The injuries evidently were not incompatible with renal function. The other three patients had a badly shattered kidney and the only course to pursue was to remove it, as a fatal abscess would have resulted if the kidney had been allowed to remain in the body.

None of the patients developed sequelae of any importance, nor did the function of the kidneys appear to be damaged in any way. The different types of posttraumatic nephritis and other renal disturbances mentioned by Wagner [26] fortunately were all absent.

In general, the prognosis appears to be favorable. The figures of other authors, cited by Wagner,[26] place the mortality in general at 18.6 per cent for all forms of treatment. The patients treated expectantly showed a mortality of 20 per cent; in those treated by conservative surgical means, which is any procedure short of nephrectomy, the mortality was 14.6 per cent, while in the cases in which nephrectomy was performed, the mortality was 16.7 per cent. There is no doubt that some of the ruptures of the kidneys that evoke symp-

toms are of slight grade and that the patients will recover without operation. It is just as certain that patients with severer renal injuries require operation and that most of them react favorably to this treatment.

PANCREAS

Subcutaneous ruptures of the pancreas are rare. Geill [1] alleged that they occur in 4.4 per cent of all visceral injuries, and most other authors concur in this view. Boesch [28] asserted, however, that the gland is frequently affected by trauma and sustains many slight contusions which are probably unrecognized during life. This assertion is hard to verify or to disprove, but, in any event, the number of pancreatic lesions that gives rise to definite complications is small.

Normally, the pancreas is placed in a retroperitoneal position with its flat surface closely applied to the posterior abdominal wall. Its longitudinal axis lies in the right and left diameter of the trunk, at right angles to the course of the abdominal aorta. Its midportion is in close proximity to the aorta and the body of the second lumbar vertebra. In front, it is covered by that portion of the peritoneum which forms the posterior wall of the lesser peritoneal sac. It is firmly anchored in its bed of adipose tissue and has no range of motion.

The structure of the pancreas is like that of other complicated racemose glands in that it is made up of small lobules separated by a vascular connective tissue which is both loose and tough. The normal organ is not easily lacerated because of its lobulated structure. Furthermore, as it lies at a considerable distance from the anterior abdominal wall, it is not easily reached by any but a violent force.

Practically the only way in which the pancreas can be ruptured is by a violence that directly compresses the lower portion of the epigastrium and crushes the viscus against the body of the second lumbar vertebra.

The object that comes in contact with the abdomen, whether it is the wheel of a vehicle, the hoof of a horse or the end of a wagon pole, is in most cases hard and consequently is well fitted to sever the pancreatic parenchyma. Perhaps the majority of ruptures are due to intense localized force inflicted by a hard object.

Boesch [28] asserted that sometimes in children whose chest wall is easily compressible a force applied to the lower portion of the sternum will force the ensiform process downward and backward, so that it meets the pancreas violently and contuses it against the spine.

The lesions produced by the violence are of different grades of intensity. The most severe type is the complete division of the gland and its peritoneal layer in the sagittal plane. Most of the lesions

28. Boesch, F.: Deutsche Ztschr. f. Chir. **167**:282, 1921.

occur at the left extremity of the gland near the tail, though a few occur at the middle of the organ or at the right extremity where the head joins the body. It is reasonable to assume that the violence would crush different portions of the pancreas, depending on whether the resistance was furnished by the central portion of the vertebral projection or the sides (fig. 4).

The usual result of the complete rupture is death by shock and abdominal hemorrhage. Some patients, however, have been operated on and have recovered. Karewski's [29] patient had a complete rupture through the middle of the gland, but was successfully treated by a

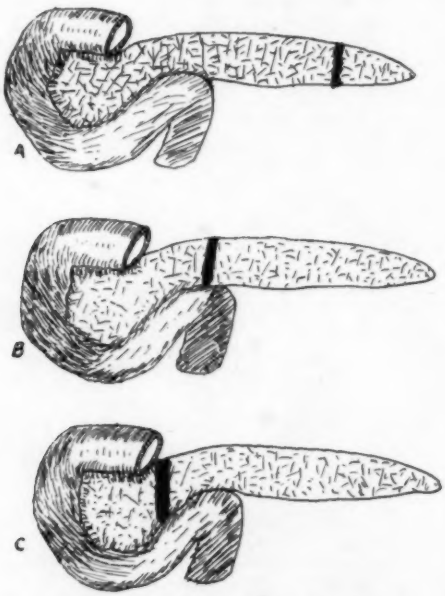

Fig. 4.—Ruptures of the pancreas.

tamponade of the injured area with a drain. Thöle [30] described a similar case. Wildegans [31] successfully sutured a complete separation of the head of the pancreas from its body, and obtained healing without after-complications. If the edges of the rupture are not necrotic, the suture of the capsule apparently is sufficient, but otherwise tamponade and drainage are necessary. The results of operation, however, are not encouraging, as the mortality is high—according to Karewski, about 72 per cent. A troublesome pancreatic fistula in the scar caused by the laparotomy frequently complicates these operations. In other instances, the lacera-

29. Karewski, F.: Berl. klin. Wchnschr. **44**:187 (Feb. 18) 1907.
30. Thöle: Deutsche Ztschr. f. Chir. **84**:45, 1906.
31. Wildegans: Arch. f. klin. Chir. **122**:276, 1922-1923.

tion of the peritoneal layer and the gland may not be extensive, and the hemorrhage may be slow. The lesser peritoneal sac will be filled with blood and, if the foramen of Winslow is in any way closed, a massive hematoma or "cystoid" will be formed. The blood will pass, however, into the general peritoneal cavity through the foramen of Winslow if that opening is patent. Occasionally, some of the pancreatic ducts are torn, and pancreatic secretion will be mixed with the blood. Small, chalky white areas of fat necrosis will be more or less widely disseminated through the peritoneal fat (Simmonds).[32] In some instances, the effused blood may become infected and a suppurative peritonitis result.

The greatest interest, however, attaches to those cases in which the peritoneal covering in front of the gland is left intact, while the pancreas and its ducts are torn more or less completely by the violence. A retroperitoneal hemorrhage, of widely varying size and position, then occurs. This finally results in that rare condition known as a traumatic pancreatic cyst.

Two forms of traumatic pancreatic cysts are distinguished: the peripancreatic cyst and the endopancreatic cyst.

The peripancreatic cyst starts to form immediately after the trauma as a huge effusion of blood and pancreatic secretion in the retroperitoneal tissues around the gland. It pushes the peritoneal investment ahead of it as it increases in size until, in a period varying from eleven days to several months, an epigastric swelling is produced that is easily demonstrable on external examination. The contents are at first bloody, but later are digested by the secretion and become clear. A reactive inflammation is set up in the periphery of the effusion, and a thick fibrous wall develops. Microscopic examination of the newly formed capsule discloses the absence of an epithelial lining for the cavity, but demonstrates the presence of necrotic gland lobules and pigment that contains iron in the wall. The clear contents of the cyst usually contain all three pancreatic enzymes.

The development of the cyst is slow and insidious. Generally, the first indications of its presence are a big fluctuating mass in the epigastrium, with progressive loss of nutrition, and numerous signs and symptoms denoting disturbance of pancreatic function, such as anorexia, constipation, glycosuria and fatty stools.

In rare instances, the cyst has eroded the wall of the intestine, and spontaneous drainage of its contents has resulted. In most cases, however, the only relief is to be obtained by operation. Tamponade and drainage of the contents have given the best results. In a few instances, complete removal of the cyst has been tried, but the pro-

32. Simmonds: Deutsche med. Wchnschr. **28**:23 (Jan. 16) 1902.

cedure is dangerous and difficult. A pancreatic fistula is prone to develop in the operative scar and may be slow to close.

The endopancreatic cysts are of two kinds: (*a*) One type is analogous to the peripancreatic variety and cannot be distinguished from it when the process is fully developed. The only difference is in the initial hematoma, which is supposed to be located more in the substance of the gland and less in the retroperitoneal layer. The development is so similar that it need not be repeated. (*b*) A second type is considered to be due to a lesion in the head of the pancreas, which finally results in the formation of scar tissue around the larger secretory ducts. An obstruction to the outflow of secretion occurs, and dilatation of the ducts consequently results. Some of the finer branches rupture, and an effusion of secretion takes place in numerous places in the gland. A reactive inflammation is set up and numerous small cysts, about 1 cm. in diameter, occur in the pancreatic substance. The process of development is slow. The traumatic etiology in this process has been vigorously disputed, as it has been described in cases in which a history of pancreatic injury was not present. Honigmann [33] and Mueller [34] described pancreatic cysts.

In the material obtained at necropsy rupture of the pancreas occurred only four times, or 4.4 per cent, in a total of eighty-nine cases of abdominal trauma. As all of these four cases were associated with severe lesions of the other viscera, not much information was to be gained from them. The clinical records did not yield any examples of pancreatic injury, thus indicating its comparative rarity.

SUMMARY

The parenchymatous organs of the abdominal cavity comprise the liver, spleen, kidneys and pancreas. They are located in the epigastrium and are well protected from external violence, so that as a rule, a marked degree of force is required to rupture them. The different organs may be injured either alone or in combination with other traumatic lesions.

The first complication that follows the accident is shock. Later on, intraperitoneal hemorrhage results from the blood vessels in the affected organ that have been torn by the rupture. In most instances, death is the result of the hemorrhage.

The other complications depend on the individual organ that has been injured. Ruptures of the liver occasionally give rise to an effusion of bile into the peritoneal cavity because of an injury to the biliary tract. Ruptures of the kidney cause trouble because of urinary

33. Honigmann, F.: Deutsche Ztschr. f. Chir. **80**:19, 1905.
34. Mueller, H.: Arch. f. klin. Chir. **143**:285, 1926.

extravasations. Injuries of the pancreas occasionally cause cysts of a peculiar type.

Ordinarily, because of the shock and the presence of blood in the abdominal cavity, injuries of the parenchymatous viscera show well marked signs of abdominal distress. In some instances the organ may be ruptured by a slight force, and the symptoms may be less outspoken. In these conditions diagnosis may be difficult, and it may be none the less difficult because external evidences of violence are often lacking.

The injuries are of different grades. A large proportion are so extensive as to be almost immediately fatal. A few are so slight that they often heal spontaneously. In between these extremes are a number of ruptures which would not necessarily result fatally if the patients were operated on and given appropriate treatment, but which would result fatally if the patients were treated expectantly.

The surgical problem is not a simple one. In a few instances, especially in ruptures of the kidney, expectant treatment may suffice. Perhaps the safest procedure is to perform an exploratory laparotomy in doubtful cases, if the condition of the patient warrants it, and then let the condition found at operation determine what course is to be pursued.

INJURIES OF THE HOLLOW ABDOMINAL VISCERA: THE STOMACH, DUODENUM, INTESTINE WITH ITS MESENTERY, AND THE URINARY BLADDER [35]

The subcutaneous injuries of the hollow abdominal viscera are unlike those of the parenchymatous organs. The former are fragile and, for the most part, poorly protected from force applied to the lower part of the abdomen. As a result, they may rupture after a slight grade of violence. The injury is often solitary and may be attended by a small amount of shock, so that the complications develop insidiously. In a large percentage of the fatal cases, death is caused by an acute suppurative peritonitis which results from leakage of the contents into the abdominal cavity. A smaller proportion of the patients die of intra-abdominal hemorrhage.

The hollow viscera are less frequently injured than the parenchymatous organs, probably because they tend to elude the action of the force. In the gastro-intestinal tract, the mobility of the segments enables them to escape the consequences of the trauma in many instances, and it is only in certain conditions that they are especially prone to rupture. The urinary bladder is likewise elusive, for in the contracted

35. Ruptures of the uterus and adnexa are not included because they are extremely rare in the normal state and usually occur as spontaneous ruptures in organs enlarged by pregnancy or pathologic conditions.

state it is well protected by the pelvis, and it is vulnerable only when it is in a state of full distention. The parenchymatous organs, on the other hand, are less free to move and cannot easily avoid the violence; even though they are better protected by their position and structure, they sustain far more injuries than the hollow abdominal organs.

THE GASTRO-INTESTINAL TRACT AND ITS MESENTERIES

Subcutaneous injuries of the gastro-intestinal tract and its membranes are relatively infrequent. Geill [1] mentioned that the intestine is ruptured in 11.1 per cent and the stomach in 7.1 per cent of his collection of cases.

There are three ways in which these organs can be affected by violence: The first method is by crushing or contusion. The force directly compresses some portion of the gastro-intestinal tube or its mesentery between the anterior abdominal wall and a hard layer of bone, like the spine or pelvis, on the posterior wall. A severe bruising of the affected portion and possibly a definite perforation of the wall may occur. At the moment of injury, the stomach or intestine contains little content, as a rule. The second method is by tearing. The force in this case is violent and compresses the anterior wall and then moves over the abdomen in a tangential fashion. Traction is put on the gastro-intestinal canal or its mesentery, and one or the other is torn in various ways. The third method is by bursting or explosion. This mechanism operates only on the stomach, duodenum or intestines and cannot act on the mesentery. It is produced by any violence which causes a sudden rise of pressure in the lumen of the tube, and the rupture results when the hydraulic force within is sufficient to overcome the resistance of the wall of the viscus. The favorable condition for the rupture is the presence of a large fluid content which distends the canal.

The most convenient way to study the gastro-intestinal injuries is to divide the tract into its natural subdivisions of stomach, duodenum and intestines. The lesions and their complications are characteristic enough for each segment to merit separate consideration.

Stomach.—Ruptures of the stomach are relatively rare. Of ninety cases of abdominal injuries, five lesions of the stomach were found. The organ lies in the epigastrium and is protected to a certain extent by its deep position and by the arch of the lower part of the chest. The violence required to damage this viscus is considerable, both because of its location and because its walls are tough and muscular. The usual trauma is severe and generalized, and other vicera usually suffer at the same time.

A contusion was described in one instance. The abdomen had been trodden on during a homicidal assault. The stomach showed symmetrical stellate ruptures of the mucous membrane on the anterior and

posterior walls, at the same level, combined with extensive contusion of the muscular coats. Perforation was not present. Death was due to an associated laceration of the liver in the sagittal plane. This case has already been described. Both injuries were produced by a severe violence which crushed the viscera between the anterior abdominal wall and the projecting lumbar spine.

Tearing of the stomach was found twice. In one case it was complete (fig. 5 *B*). A severe tangential violence traveling across the

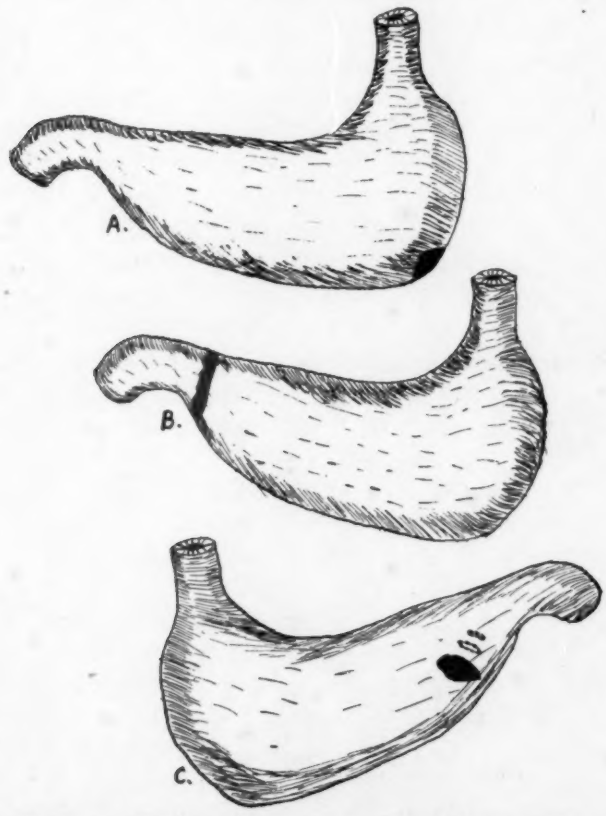

Fig. 5.—Diagrams showing different ruptures of the stomach. *A*, Bursting rupture of the stomach. *B*, Complete tear of stomach at pylorus. *C*, Partial tears of stomach near pylorus on posterior wall.

epigastrium from right to left had put traction on the pylorus, tearing it transversely across 1.5 cm. from the firm duodenal attachment. In the other instances, the tear was partial. The lesion was an elliptic perforation of the posterior wall midway between the pylorus and fundus (fig. 5 *C*). This was combined with numerous smaller tears on the mucous and serous surfaces. All indicated the action of a force that passed across the epigastrium from right to left. Because of its

exposed position, the pylorus is much more vulnerable to the tearing mechanism than the more deeply placed cardia.

A bursting or explosive rupture of the stomach was encountered twice. The organ was distended with food content at the moment of the casualty and, when pressed on, ruptured at the fundus. The lesion appeared as a small circular perforation, from 1 to 2 cm. in diameter, situated on the most dependent portion of the greater curvature, somewhat to the left of the median line of the abdomen (fig. 5 *A*). The stomach was contracted, and the abdomen was full of acid food content and brownish blood.

In these cases, death was due to shock and hemorrhage from injuries of other viscera, though in two cases the acid gastric content had set up a marked chemical peritonitis, which contributed to the death of the patient.

Duodenum.—The duodenum is more deeply placed, but is occasionally ruptured because of the greater fragility of its walls. It is a U shaped tube situated in the epigastrium around the head of the pancreas. Its more distal portion passes over the bodies of the lumbar spine, and it is at this point that a severe localized violence can impinge on its wall. A case is described in which the duodenum was crushed between the anterior abdominal wall and the vertebral bodies and sustained a complete perforation of the anterior wall and a partial perforation of the posterior wall, at the same level (Vance).[36] Tears also occur at this point and are caused by a tangential force which puts a transverse traction on the most dependent portion of the tube, severing it either partially or completely. Bursting ruptures are found and are the probable result of a force applied directly to the duodenum when it is in a state of distention. The lesion appears as a circular opening in the most dependent portion of the U shaped canal.

The result of the perforation is to allow the contents to escape into the retroperitoneal tissues, with the consequent production of a gaseous gangrenous cellulitis which proves speedily fatal. This complication may give definite clinical signs which may lead to an exploratory laparotomy. Unfortunately, because of its retroperitoneal location, the lesion is frequently overlooked at operation (Berry).[37]

Intestines.—The intestines are injured more frequently than the stomach or duodenum, principally because they are placed in an exposed position in the abdominal cavity. The small intestine is arranged in coils and fills a space from a level just above the umbilicus down to

36. Vance, B. M.: Traumatic Lesions of Intestine Caused by Nonpenetrating Blunt Force, Arch. Surg. **7**:197 (July) 1923.

37. Berry: Brit. M. J. **2**:643 (Oct. 22) 1921.

the pelvic floor. It is protruded against the anterior abdominal wall by the projection of the lower end of the lumbar spine. The large intestine is less exposed. The ascending colon, the descending colon and the sigmoid are placed well back in the lumbar fossa. The transverse colon crosses the front portion of the abdomen, but it is vulnerable only at the midline of the trunk, where it may be crushed between the anterior abdominal wall and the bodies of the lumbar vertebrae. For this reason, the injuries of the large intestine are much fewer than those of the small intestine. Berry described 177 injuries of the small intestine and 15 of the large intestine.

As the tears of the intestines are frequently produced by the same mechanism that tears the mesentery, it is convenient to consider them together. The resulting lesions depend on the way in which the violence is applied. A force that pulls the intestine in the direction of its long axis may snap it completely across, and then may continue the tear into the mesentery, producing a slit that runs toward the base of the membrane.[30] Traction exerted in the line of the vascular supply of the mesentery, away from the base and across the intestinal tube, tends to tear large ovoid spaces in the membrane and may crush or cause a partial tear of the intestinal wall. Partial tears can occur from traction applied either in the long axis or in the transverse axis. Needless to say, all varieties and combinations of tears occur.

Seven tears of the small intestine were found in the material obtained at necropsy, five of which were partial and two complete. The accidents were caused by automobiles in four cases; street cars in two cases, and a blow from a falling stone in one case. The complete tear is found for the most part in the upper portion of the jejunum, a few inches distal to the ligament of Treitz, where the intestine is firmly fixed to the posterior abdominal wall. Immediately after the trauma, the circular muscle in the severed ends firmly contracts and prevents leakage of the contents into the peritoneal cavity. After several hours have passed, the muscle relaxes and the contents escape. Death occurs either in the early stages from hemorrhage from the torn mesentery, or in the later stages from peritonitis, which is almost certain to follow leakage of the contents.

Partial tears are frequently seen as large, ragged, ovoid perforations on the peritoneal surface of the intestine. Often the serous layer is stripped in a zone around the hole through the muscular layer. In one case of this kind, an *Ascaris* worm was present in the intestine near the perforation and had been severed by the violence (fig. 6).

Contusions occur in various forms and may be of all grades of severity. If the force is a localized one with a small area of impact, the intestine may be perforated by the direct violence and may show

marked ecchymosis around the lesion. The bruising may injure the wall to such an extent that a secondary perforation may occur a few days after the injury and cause a fatal peritonitis. In a few instances, the injury can cause a necrosis of the wall and so damage the vitality of the tissues that bacteria will enter the peritoneum without actual perforation.

Contusions of the mesentery occur, but as they are rarely associated with intestinal injuries, they will be discussed later.

Fig. 6.—Partial tear of small intestine with injury to an *Ascaris* worm in that segment.

Injuries resulting from bursting occur, and their appearance is characteristic. They are found as small circular or ovoid perforations on the wall, generally opposite the mesenteric attachment but occasionally near it. Most of them are single, but in a few instances they may be multiple. They are due to some force that causes a rise of pressure inside the cavity of the intestine, which at the time of injury is distended by fluid. Different explanations have been advanced to account for this mechanism.

Sauerbruch [38] asserted that the force imprisons a coil, prevents its contents from escaping and causes the rupture by a sort of hydraulic action. Haim [39] said that any sudden contraction of the abdominal cavity, whether due to violence or even to muscular action, will cause a rise of pressure inside the intestine and that bursting will occur where the wall is weakest. The presence of a hernial sac always aids in the production of this lesion, as the coverings of the sac are too weak to support the intestinal wall against the explosive force within its lumen.

Spontaneous ruptures of the gastro-intestinal tract are of the bursting variety. Any process that weakens the wall, such as an ulcer or an area of necrosis, will predispose to the perforation. This condition, however, belongs to the category of an erosion rather than that of rupture. The best example of a spontaneous rupture, perhaps, is to be found in intestines incarcerated in hernial sacs, in which the vitality of the wall has been so diminished that even mere attempts at reduction cause an explosive perforation.

A study of the material obtained at necropsy (including twelve cases reported by Vance [36]) showed a total of twenty-four cases of ruptured intestine. The duodenum was involved in three cases and the small intestine in twenty-one. The three duodenal injuries were caused by contusion, tearing and bursting. The injuries of the small intestine were caused by contusions in two cases; tearing in seven cases, and bursting in twelve cases. In this list the ruptures caused by bursting outnumbered the contusions and tears combined, which suggests that such ruptures are certainly more common than has hitherto been supposed. As they can be produced by a slight trauma, the numerical preponderance of this type is a matter of some importance.

The trauma was generally a slight localized violence applied to the lower part of the abdomen. The casualties were caused by: automobiles in one case; kicks in the abdomen in two; falls in three; a thrown missle in two, and an unascertained injury of slight grade in four. An inguinal hernial sac was present in six of the twelve cases and undoubtedly favored the production of the lesion.

Injuries of the large intestine and appendix were not found. In one instance a mucocele of the appendix had been burst by a slight trauma, and the abdominal cavity contained a handful of clear jelly-like mucin. The appendix, however, was markedly distended and the rupture was really spontaneous.

Reports of ten clinical cases were collected, five in which death occurred and five in which recovery took place.

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38. Sauerbruch: Mitt. a. d. Grenzgeb. d. Med. u. Chir. **12**:92, 1903.
39. Haim, E.: Arch. f. klin. Chir. **93**:685, 1910.

CLINICAL HISTORIES

FATAL CASES

CASE 1.—A man, aged 65, fell downstairs, landing on his abdomen. He was admitted to the hospital twenty-two hours later in a state of shock, complaining of pain, distention of the abdomen and vomiting. He had bilateral inguinal hernial sacs, the one on the left being the larger. A laparotomy was performed shortly after admission. A perforation of the ileum, 1 cm. in diameter, was present, about 3 feet (91 cm.) proximal to the ileocecal valve. This was closed by suture. Death occurred about a day and a half after the injury.

CASE 2.—A boy, aged 13, fell from a chair, landing on his abdomen. He complained of severe abdominal pain, vomiting of feculent material and diarrhea. He was admitted to the hospital thirty hours after the injury in a marked state of shock, with abdominal pain and distention. The leukocyte count was 44,000, and the polymorphonuclears numbered 94 per cent. A laparotomy was performed shortly after admission. A perforation 1 cm. in diameter was found near the mesenteric insertion of the jejunum, in the upper portion. A fully developed peritonitis was present. The perforation was closed by suture. Death occurred about two days after the trauma.

CASE 3.—A boy, aged 7, was knocked down by an automobile. He was admitted to the hospital six hours after the injury, complaining of abdominal pain, tenderness and intense thirst. The urine was moderately hemorrhagic. Abdominal rigidity was marked. The preoperative diagnosis was "rupture of bladder and fracture of pelvis." A laparotomy was performed shortly after admission. Three perforations of the intestine were found and sutured. The patient died the next day in collapse.

CASE 4.—A man, aged 46, was injured in a trolley car collision. He complained of pain in the right lower quadrant of the abdomen, which steadily grew worse. He was admitted to the hospital two days after the accident in severe shock and with marked abdominal distention. A laparotomy was performed shortly after admission, disclosing a fully developed purulent peritonitis. The jejunum showed a perforation on the side opposite the mesentery, 2 cm. in diameter. An enterostomy was performed near the perforation. The patient died three days after the accident.

CASE 5.—A man, aged 21, was crushed between two cars. He was admitted to the hospital in a marked state of shock and with a painful and tender abdomen. A laparotomy was performed shortly after admission. A complete cross tear of the jejunum was found, with a 4 cm. mesenteric tear at a point 40 cm. below the ligament of Treitz; 40 cm. below this were two areas in which about 15 cm. of the small intestine were free of mesentery. A partial enterectomy and an entero-enterostomy were performed. The patient died after two days of an acute suppurative peritonitis.

CASES IN WHICH RECOVERY TOOK PLACE

CASE 6.—A man, aged 26, was injured in a collision of a taxicab with a wagon. He was admitted to the hospital immediately after the injury, with sharp pain in the left side of the abdomen and an abrasion over the left flank. Laparotomy was performed shortly after admission. A perforation of the small intestine, 1 cm. in diameter, was found. This was closed, and drainage was instituted. After operation, the patient passed much gas by rectum for

four days. The drainage tract discharged foul material for about ten days. The patient fully recovered within one month after operation, without sequelae.

CASE 7.—A man, aged 29, was struck in the right lower quadrant of the abdomen by a piece of wood thrown from a circular saw. He was admitted to the hospital complaining of pain over the abdomen. A laparotomy was performed shortly after admission. The abdominal cavity contained much content from the small intestine, with an abundant fibrinous deposit on the serous surface. About 2 cm. from the cecum was a small perforation of the ileum, 3 mm. in diameter. This was closed by suture. A jejunostomy was performed, and a catheter was inserted through an artificial opening because of the distention. A toilet of the peritoneum was performed. The patient was very ill for the first few days after the operation, but recovered in one month without sequelae.

CASE 8.—A man, aged 36, was crushed by a trolley car. On admission to the hospital, he was in a state of shock, with evidence of fractured ribs on both sides and a rigid and distended abdomen. A laparotomy was performed immediately. An oval opening, about 1.5 by 1 cm., in the ileum was found and sutured. After a stormy convalescence from an injury in the right side of the chest, the patient recovered in forty-three days.

CASE 9.—A man, aged 59, while lifting a barrel of apples, was suddenly seized with severe knifelike pains in the abdomen. He was admitted to the hospital with rigidity and tenderness over the abdomen. A laparotomy was performed several hours after admission. Much turbid brown fluid was found in the abdomen, and a definite fibrinous peritonitis was present. A perforation of 1 cm. of the small intestine was discovered and sutured. An enterostomy was performed with a catheter about 15 cm. proximal to the perforation. An uneventful recovery occurred after thirty-eight days, without sequelae.

CASE 10.—A man, aged 52, was crushed between a truck and a box car. He was able to walk after the accident. He was admitted to the hospital complaining of severe cramplike pain near the umbilicus and much abdominal distention. An incarcerated scrotal hernia was found on the right side. A laparotomy was performed shortly after admission. The ileum was completely divided with a laceration 4 cm. long into the mesentery which was situated about 10 cm. from the ileocecal valve. Much blood and fecal material was present in the abdomen and was removed by suction. Paul tubes were put in the severed ends of the small intestine. Eight days after the operation, the two ends of the intestine were closed by suture and a drain was put in. Recovery was slow and lasted fifty-eight days. The fistula was at first fecal, but cleared up during the first month after discharge. A ventral hernia persisted, however.

These cases illustrated the usual clinical signs of the ruptured intestine. In general, they showed a moderate grade of shock, with well marked signs of peritoneal irritation which grew progressively worse. Pain, tenderness, distention and rigidity of the abdominal wall were the chief indications of the rupture. External wounds of the abdomen were not present, and in only two instances were signs of any external trauma present on the body.

Of the ten patients, five died and five recovered. The patients who died varied in age from 7 to 65 years. They had suffered casualties

of varying grades of severity. Patients 3 and 5 showed more or less shock after the injury; the laparotomy was done shortly after the trauma, but apparently was not of much avail. Patients 1, 2 and 4 were admitted anywhere from twenty-two to forty-eight hours after the trauma, and a fatal peritonitis was fully developed when the operation was started. The lesions consisted of bursting ruptures in four instances and tearing of the intestine and mesentery in one.

The five patients who recovered varied in age from 26 to 59 years. Some of the injuries were slight; others were severe. The patients were admitted, however, soon after the accident and were operated on within a few hours of their admission. Four of the injuries were bursting ruptures and one was a tear completely through the ileum Some of the patients showed intestinal contents free in the abdominal cavity at the time of operation, and signs of peritoneal inflammation were present.

Apparently the important factor in treating patients with intestinal injuries successfully is to perform the operation early. Unfortunately, the seriousness of his condition is not always recognized by the patient, and he may not seek medical attention until he is practically moribund. Then, too, because of the vagueness of the early symptoms and the frequent absence of external evidences of violence, the surgeon is prone to overlook the possibility of an intestinal lesion. In several instances, alcoholic intoxication at the time of the accident served to mask the signs of the abdominal trouble. The result is that some patients who might have been saved by prompt surgical measures die because of the delay.

The usual surgical measure is to open the abdomen and close the opening in the intestine. If the perforation is small, this is not difficult. With large openings and mesenteric tears, resection of the intestine around the lesion is sometimes necessary. In cases of severe ileus, an enterostomy is frequently necessary to combat the distention of the intestine.

Mesentery.—The mesentery is occasionally ruptured subcutaneously, either alone or in combination with the intestine. Two mechanisms have been described: (1) contusion and (2) tearing.

1. In rare instances, a localized violence with a sharply limited area of impact can thrust against the anterior abdominal wall at right angles and crush the membrane against the iliac bones or the lower end of the lumbar spine (Dubs).[40] If a single layer is involved, a single perforation is produced. If several folds of the membrane are caught, several perforations result. The folds nearest the abdominal wall generally show an opening with ragged and contused edges, about the

40. Dubs, J.: Deutsche Ztschr. f. Chir. **151**:120, 1919.

size and shape of the object that has caused the trauma. The other folds show perforations of somewhat smaller diameter, and the one farthest away from the point of violence may be lacerated only on the surface that happened to be presented anteriorly. With this mechanism, a mesentery heavily interlarded with fat is more prone to rupture than one that is thin and lax.

The ruptures represented in figure 7 were in all probability produced by this type of violence, though a complete history of the casualty

Fig. 7.—Perforations of the mesentery of the small intestine due to a severe contusion.

could not be ascertained. A man, aged 45, was found unconscious on the sidewalk. On admission to the hospital, a diagnosis of acute alcoholism was made, and the presence of an abdominal injury was not suspected. The patient died nine hours afterward. At necropsy, the abdominal cavity was found full of blood and blood clot; the mesenteric lesions were also discovered and were obviously the source of the hemorrhage. An area of contusion was also present in the fat of the anterior abdominal wall, just above the umbilicus, and also on the posterior

abdominal wall, just to the inner side of the cecum. The anterior surface of both thighs presented considerable bruising, and a contusion was present on the back of the right hand. It was obvious that the man had been in an accident and had probably fallen heavily against a projecting object.

2. More commonly, the mesentery is torn by an indirect violence which acts tangentially on the abdominal wall and exerts traction on the membrane. This mechanism had already been considered in the tears of the mesentery and intestine combined. The same force can act on the membrane alone and can produce either huge isolated ovoid openings or multiple smaller openings. All grades of the lesion occur. Complete tears involving both layers are the most common, but partial rips, which involve only one side of the membrane, have been seen. Both may be present in the same case. Occasionally, the intestine may be stripped from the mesenteric attachment, as described by Dubs.[41]

The casualty that is most commonly responsible for this lesion is the highway accident. The wheel of an automobile grinds the body and exerts traction on the membrane. Less often, a localized violence which ploughs its way over the anterior abdominal wall will produce a similar effect. Figure 8 shows an example of this type of force. A man, aged 33, while seated on his truck, ran into the end of a projecting pole from another vehicle and forced it tangentially over his abdomen.

The mesentery of the small intestine is most often injured, especially at the cecal end. Less often, the mesosigmoid, the mesocolon transversum, the omentum and the meso-appendix are involved. The usual result is an intra-abdominal hemorrhage. If the big vessels at the root of the mesentery are torn, the bleeding is rapid and is followed by death in a few minutes. More often the bleeding is slower, and the hemorrhage may be prolonged for hours and even days. An associated intestinal perforation will result in a fatal septic peritonitis.

Another complication is introduced when the rupture occurs adjacent to the intestinal insertion. Here the arterial vessels are end arteries, so that a mesenteric injury of any size will interfere with the blood supply of the intestine at this point. If death does not occur from hemorrhage in the course of one or two days, necrosis and gangrene of the affected intestine will result, with the further development of a severe toxic and paralytic ileus, which finally ends in death. The following case illustrates this complication:

A man, aged 36, fell during the collapse of an excavation under a sidewalk. He did not enter the hospital until the second day after the accident and was then suffering from severe abdominal tympanites and was practically moribund. A rapid enterostomy was performed to relieve the distention, but

41. Dubs, J.: Deutsche Ztschr. f. Chir. **133**:366, 1915.

without avail. The patient died two days later. At necropsy, a perforation of the mesentery, 6 cm. in diameter, was found adjacent to the ileum, about 20 cm. from the cecal junction. The intestine adjacent to the perforation was dark and gangrenous, as indicated in figure 9. That portion of the small intestine distal to the injury was contracted, while the proximal portion was massively distended and in a state of acute ileus. The abdominal cavity contained about a pint of discolored blood and blood clot. Death was obviously the result of the toxic ileus.

Cases have been described in the literature in which a mesenteric rupture of this sort, too small to produce gangrene, has caused a chronic

Fig. 8.—Multiple tears of the mesentery of the small intestine.

stenosis of the intestine after a lapse of several months. The process seems to be dependent on the fact that the blood supply of the stenotic area has been curtailed by the lesion in the mesentery. An inflammation of the connective tissue takes place in the intestine, causing fibrosis and shrinking of the wall and narrowing of the lumen to a small caliber. A chronic obstruction is the result, which can be remedied only by resection of the stenotic area (Dubs [40] and Maier).[42]

In the material obtained at necropsy, only seven cases out of ninety, or 9.7 per cent, showed mesenteric tears that were not associated with

42. Maier, O.: Arch. f. klin. Chir. **132**:212, 1924.

another serious visceral injury which could be said to have caused death. The ages varied from 3 to 60 years. Four patients died rapidly, within three hours, of an intra-abdominal hemorrhage; two died of abdominal hemorrhage nine and ten hours, respectively after the accident; one died at the end of four days from the associated gangrene of the ileum and its sequelae, which have already been described.

Three cases showed solitary tears in the mesentery at the lower end of the ileum. The other four tears were multiple and involved the

Fig. 9.—Perforation of the mesentery of the small intestine with gangrene of the adjacent portion of the intestine.

mesentery of the small intestine in all instances and the omentum in three instances. The violence was severe in all cases. Five of the tears were caused by accidents on the street, one from a fall down an excavation, while the other was probably caused by a fall.

An isolated mesenteric rupture was not discoverable in the clinical material, though two tears were found in the ten cases already listed under intestinal injuries, in one of which the outcome was fatal and in the other of which the patient recovered. Because of the intra-abdominal hemorrhage, the mesenteric injuries were outspoken in

their abdominal manifestations, though, as has been shown, this does not necessarily occur in all cases. They present the same difficulties of diagnosis that have been noted in other abdominal injuries.

The only treatment for patients who have these lesions is to operate, tie the torn vessels and, if necessary, resect adjacent portions of the intestine so as to prevent the onset of gangrene in that area. Expectant treatment is of no avail.

The Urinary Bladder.—Subcutaneous ruptures of the urinary bladder are described as being relatively rare. Geill [1] found only 4.4 per cent in his collection of reports of visceral injuries.

The anatomic position of the urinary bladder in the abdomen and the changes in relationship to the surrounding structures that it undergoes during contraction and distention explain many peculiarities of ruptures of the bladder. In the fully contracted state, it lies entirely extraperitoneally and is fully protected by the strong bony ring of the pelvis. In this state it is most often injured by blunt force which fractures the pelvis. As the bladder fills with urine, it projects above the pelvic ring and finally, in a condition of full distention, occupies most of the lower part of the abdomen and is invested by peritoneum on its posterior and superior surfaces. Its fundus is placed between the promontory of the sacrum and the anterior abdominal wall. In this position, it is especially vulnerable to any violence that may be applied to the lower part of the abdomen.

The ruptures of the bladder are classified most conveniently as: (1) extraperitoneal and (2) intraperitoneal.

1. The extraperitoneal ruptures are of two types: those associated with fractures of the pelvis and those which occur without pelvic fractures. The first subdivision is the more common and comprises lacerations inflicted on the wall of the bladder by the sharp ends of the broken pelvic bones. The location of the lesion depends entirely on the site of the fracture. At the time of injury, the bladder either is empty or contains a small quantity of urine.

A common location for an extraperitoneal tear is on the anterior wall near the midline, half-way between the fundus and the urethra. The violence first causes a fracture of the pubic rami and depresses the sharp ends of the broken bones backward. The sharp inner fragment of the superior ramus lacerates the anterior surface of the viscus, and a big ovoid hole into the cavity is the result.

A second site of these ruptures is usually on the posterior lateral wall, a few centimeters above and behind the ureteral orifice, immediately in front of the corresponding sacro-iliac articulation. When this joint is luxated by the violence, a forward projection of the sharp sacral segment will generally perforate the bladder in this region.

In some fractures of the pelvis, all four pubic rami are fractured and the entire pubis is detached from the rest of the pelvis. If this is forced backward by the violence, the membranous urethra will be torn across close to the prostate.

All three varieties of laceration may occur, either isolated or in combination, depending on the way in which the fracture is produced.

The force required to cause these injuries is necessarily violent, as the pelvis is not easily broken. Consequently, a severe shock results, and the patient often dies of this complication within a few hours of the accident. In some instances the shock is not so severe, and then an extravasation of blood and urine occurs through the lacerations in the wall of the bladder and may reach voluminous proportions. It may fill the perivesical retroperitoneal tissues, run down into the thighs, the scrotum, the perineum, the buttocks or the anterior abdominal wall. The end-result of the process is a gangrenous abscess of the parts involved that is apt to prove fatal through septic infection.

In rare instances, an extraperitoneal rupture may occur without a pelvic fracture. At the moment of trauma, the bladder is in a state of half distention and is acted on by a force that presses on the lower abdominal wall from above downward. The impact is against the fundus and the increased pressure within the lumen is directed most strongly against the base of the organ. The bladder tends to rupture at the various points of lessened resistance in this region, which are the great sciatic foramina, the obturator foramina and the pelvic outlet (Schonwerth).[43] One case of spontaneous rupture in this region, complicating a chronic urinary obstruction due to prostatic hypertrophy, has been described (Muir).[44]

2. The intraperitoneal rupture occurs only when the bladder is fully distended by urine. A force applied to the anterior abdominal wall below the umbilicus will compress the posterior part of the fundus against the promontory of the sacrum and raise the pressure inside the lumen. The apex of the sac is thus put under stress and consequently ruptures. In most instances the perforation is found as a longitudinal, ovoid opening, slightly posterior to the apex. Some perforations, however, may be located laterally or even anteriorly. The violence is often slight and, as a rule, the bladder is the only organ injured. In some cases, even indirect violence, such as a fall on the buttocks or a sudden increase in intra-abdominal pressure from muscular action such as lifting a heavy weight, can cause the same lesion (Seldowitsch).[45]

A large number of spontaneous ruptures at the fundus have been described. In the majority of instances, a predisposing cause has

43. Schonwerth, A.: Arch. f. klin. Chir. **85**:876, 1908.
44. Muir: Brit. M. J. **2**:25 (July 4) 1908.
45. Seldowitsch, I. B.: Arch. f. klin. Chir. **72**:859, 1904.

weakened the bladder wall, such as chronic obstruction to the outflow of urine referable to prostatic hypertrophy or to an old urethral stricture. In other cases, the wall of the bladder may be definitely weakened by a neoplasm, tuberculosis, lipomatosis, ulceration of any sort or trophic changes that result from diseases of the central nervous system. In a state of distention, rupture can occur from such slight stress as would be furnished by a sudden movement or straining at stool (Dittrich).[46]

Occasionally the intraperitoneal rupture is associated with a fracture of the pelvis, though this is merely an accessory action of the violence and has no direct effect on injury of the bladder. Two cases of this sort were noted, both the result of injuries received in automobile accidents. In one the left acetabulum was broken, and in the other the pubic bones were separated at the symphysis. The bladder in the latter case is shown in figure 10.

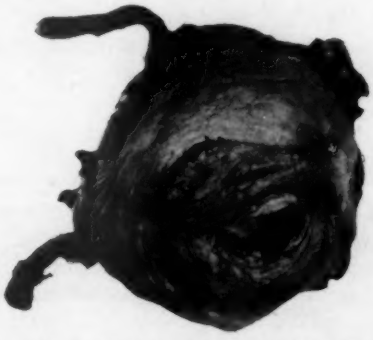

Fig. 10.—Intraperitoneal perforation of the fundus of the urinary bladder.

At necropsy the bladder is found markedly contracted, with the opening at the fundus plainly visible. The abdomen is generally full of urine, which is sometimes clear and sometimes tinged with blood. In a patient who died shortly after the trauma, the serous surface may be unchanged. If from two to three days have elapsed, however, the peritoneum is generally reddened and congested and shows a low grade inflammatory reaction. According to Rost,[47] much of the urine is absorbed from the abdominal cavity, and the patient may die of uremic symptoms. In other instances, bacteria may gain entrance by way of the perforation in the bladder, either from urine primarily infected or from the injudicious introduction of a contaminated catheter into the bladder. The result in that event is death by acute suppurative peritonitis.

46. Dittrich, R.: Deutsche med. Wchnschr. **48**:974 (July 21) 1922.
47. Rost, F.: München. med. Wchnschr. **64**:5 (Jan. 2) 1917.

A few cases are described in which the intraperitoneal opening has been closed later, so that the escape of urine into the abdomen was prevented. In one case it was noted that a piece of omentum had been snared in the rupture and had sealed the orifice. In other instances the perforation of the bladder is directed obliquely through the wall, so that the outflow of urine is prevented by a valve action (Nordmann).[48]

Few cases came to necropsy, and, as most of them have been discussed, further detail will not be given.

The clinical records follow.

CLINICAL HISTORIES

CASE 1.—A man, aged 44, fell to the floor the night before admission while in an alcoholic condition. His abdomen began to swell; he noticed that he could not void urine and vomited considerably. He was admitted about twelve hours after the fall, complaining of abdominal distention and vomiting. The abdomen was tympanitic, with signs of fluid in both flanks. Catheterization yielded a straw-colored urine. He was operated on about ten hours after admission. The abdomen contained from 5 to 6 liters of turbid yellow fluid. A rupture of the bladder, 4 cm. long, was found on the posterior part of the fundus. It was sutured, and the patient died two and one-half days later from toxemia.

CASE 2.—A man, aged 36, was crushed in an elevator. He was admitted to the hospital in a state of shock, showing multiple contusions of the scalp, pelvis and lower part of the abdomen. Pain and tenderness in the lower part of the abdomen, with some rigidity, were present. He was operated on the day after admission. Clear, colorless, odorless fluid was found in the abdomen, and a rupture of the bladder was found on the posterior portion of the fundus, which was sutured. The appendix appeared inflammed and was removed. The patient died two days after the trauma.

CASE 3.—A woman, aged 24, jumped from a third story window while under the influence of alcohol. She was admitted to the hospital the day after the accident in a state of shock and complaining of severe abdominal pain. The abdomen was tympanitic and distended. She said that she could not void. Only a few cubic centimeters of bloody urine were obtained on catheterization. There was a fracture of the right iliac bone and the left thigh, and numerous contusions and lacerations were present over the face, extremities and trunk. An operation was performed two days after the injury. Two quarts (946.2 cc.) of straw-colored fluid with a urinous odor was found in the abdominal cavity. The peritoneum was congested, but did not show an exudate. A tear, 2.5 cm. long, was found in the fundus of the bladder, which was closed. A suprapubic cystostomy was performed, and a drain was placed between the bladder and the uterus. Recovery was slow, due to the broken bones. The patient was discharged at the end of the second month with only the sequelae due to the broken bones.

In spite of the small number of cases, several points of interest were brought out. One was the association of alcoholic intoxication with

48. Nordmann, O.: Deutsche med. Wchnschr. **34**:144 (Jan. 23) 1908.

the trauma, which was most typically illustrated by case 1. Apparently a slight force is sufficient to rupture a distended bladder in a patient under the influence of alcohol. This was emphasized long ago by Bartels,[49] who not only mentioned that the beverage tended to fill the bladder with urine, but also said that it even tended to weaken its musculature, so that it was less resistant to slight stress. In addition, the anesthetic action of the alcohol would probably render the abdomen insensitive to irritation and delay the onset of typical symptoms. For this reason, many patients would probably delay seeking medical attention.

All the cases displayed the signs of abdominal distress which would naturally follow the introduction of a slightly irritative fluid in large quantities into the abdominal cavity. The inability to void urine was noted in two cases, and the yield of a small amount of blood on catheterization was noted in one. These signs are given great prominence in the literature as important indications of a vesical perforation.

The escape of urine into the abdomen apparently is not so serious as the escape of intestinal contents. Patient 3 was operated on about one and one-half days after trauma and went on to final recovery. The urine seemed to cause, at the most, only a slight irritation of the peritoneal surface. However, the most advisable course after rupture of the bladder is an immediate laparotomy and repair of the perforation, whether it is intraperitoneal or extraperitoneal. The continued extravasation of urine may lead to complications which are apt to prove fatal.

SUMMARY

The hollow abdominal viscera comprise the gastro-intestinal tract plus its mesentery and the urinary bladder. They are located mainly in the lower portion of the abdomen, where they are more or less exposed to trauma. They can be readily ruptured by a moderate force applied to the anterior abdominal wall, so that in many instances they may display few symptoms immediately after the injury.

The usual complications which develop after the trauma are hemorrhage into the abdomen, usually from a torn mesentery, or a peritonitis of varying grade from the extravasation of urine or intestinal contents into the peritoneal sac.

The only method of cure in these cases is by operation. Any injury of a hollow viscus, if capable of admitting its contents into the abdominal cavity, must be taken seriously, and the lesion should be repaired as soon as possible. Intestinal ruptures especially are more virulent than ruptures of the urinary bladder, because the contents of the former are more infective. Early operation, in any event, offers the only chance of recovery.

49. Bartels, M.: Arch. f. klin. Chir. **22**:715, 1878.

THE ETIOLOGY AND TREATMENT OF SCOLIOSIS *

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The importance of early and efficient treatment of patients with scoliosis, arises from the extreme uncertainty of their future progress. It is known that in some patients the condition may become spontaneously arrested in an early stage; in other cases, the disease may increase, and may not only produce extreme deformity but seriously impair the general health. The aim of treatment should be to prevent the progress of the deformity, to correct, if possible, the existing deformity and to maintain the correction until all danger of relapse is past. This usually means until maturity is reached.

The correction of spinal curvature requires much attention to detail, and individual adaption of the various procedures, varying largely with the location and degree and, to a lesser extent, with the etiology of the deformity. For this reason it was thought desirable to study a series of cases to determine, as accurately as possible, the predisposing and direct etiologic factors and associated conditions, and to compare the results of treatment by various methods.

As a basis for this study, the clinical records and roentgenologic observations in 125 cases of scoliosis were reviewed These cases were selected from those observed between the years of 1919 and 1927 by my colleague Dr. Willis C. Campbell and myself.

Thirty-six of the patients, observed within the past three years, have been studied with particular reference to the results of treatment.

AGE AT ONSET

One hundred and twenty of the patients were white and five were colored; eighty-seven, or 70 per cent, were females and thirty-eight, or 30 per cent, were males. The average age at which the deformity was first noticed was 12½ years in both male and female patients. The average age at which the physician was first consulted, however, was 14 years for girls and 18 years for boys. This means that parents are more concerned about good figures for their girls than for their boys. Ten patients were under 10 years of age; when first examined the youngest was a child of 16 months with a congenital malformation of the spine. Ninety-seven were in the second decade of life; fifteen in the third decade; two in the fourth decade; and one, the eldest, was a woman of 68, who had had a curvature of the spine as long as she could remember and who sought treatment for rheumatoid arthritis.

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* From the Willis C. Campbell Clinic, Memphis.

Obviously, the age at onset is difficult to determine, because in a majority of cases the deformity was discovered accidently at a time when it was well developed, and it was impossible to estimate how long it had been present. In the case of girls, scoliosis is often first noticed by the dressmaker, or when examination of the back is made following some minor injury. In a fairly large proportion of our cases the spinal deformity was discovered during a routine examination by the family physician or during examination at school or at a scout camp. This is gratifying, as it indicates that the medical professon as a whole is becoming informed of the importance of the condition, is realizing that the child will not outgrow the deformity and is insisting on proper and efficient treatment.

SYMPTOMS

The symptoms vary somewhat with the etiology. They are mild at onset, and often the deformity is the only complaint. This, however, is usually progressive. In the idiopathic group, the children are often tall and slender, slightly undernourished and growing rapidly. They may complain of tiring easily on exertion and of having a dull backache, which is at times referred to the pelvic or shoulder girdle. There may be awkwardness in walking, shortness of breath and nervousness. Cardiac irregularity and tacchycardia were noted in a few cases.

ETIOLOGY

The etiology of scoliosis, especially of the static or idiopathic variety, is obscure. According to Lovett,[1] it originates in the flexed position of the spine, and the immediate cause is some asymmetry of development or posture which leads to the superincumbent weight of the upper part of the body being borne in an oblique direction on an unstable spine, thus causing it to deviate from the midline. Any faulty attitude habitually assumed by the growing child will result in uneven distribution of the weight and is the direct cause of the deformity.

The predisposing factors are varied, and many theories have been suggested. For example: (*1*) It has been claimed that all cases are due to unrecognized anterior poliomyelitis which may have involved only the spinal muscles. (*2*) Redard [2] suggested the large incidence of flatfoot coincident with scoliosis as an etiologic factor. As will be shown later, this does not hold true in our series. (*3*) Brackett,[3] Scud-

1. Lovett, Robert W.: The Mechanics of Lateral Curvature of the Spine, Tr. Am. Orthop. A. **13**:251, 1900.

2. Redard, P.: De la scoliose dans ses rapports avec le pied plat, Tr. Am. Orthop. A. **9**:339, 1891.

3. Brackett, E. G.: An Etiological Factor in Lateral Curvature, Tr. Am. Orthop. A. **9**:207, 1896.

der [4] and others have emphasized the laxity of the spinal ligaments, the muscular weakness and the decreased density of bone. (*4*) Lee [5] accounted for the high incidence of the condition in girls at puberty as being due to stimulation of the sacral plexus by pelvic congestion. (*5*) In the series of cases reported by Adams,[6] there was a large percentage with congenital variations of the lumbosacral spine.

In this series the etiologic factors which were chiefly responsible for the deformity were in the order of frequency: idiopathy, seventy-nine cases; anteriopoliomyelitis, twenty-five cases; congenital anomaly of the bones of the spine, ten cases; empyema, four cases; spastic paralysis, three cases; destructive lesion of the vertebra, two cases; rickets, one case, and torticollis, one case.

The cases have been classified in this manner when in the opinion of the observer some etiologic factor was the chief or only factor involved. Twenty-eight cases, however, presented a combination of more than one possible cause. Twenty-four of these cases, on roentgenologic examination, showed some minor congenital spinal anomaly which in itself was not deemed sufficient to account for the deformity. This will be discussed further in the section under congenital scoliosis. Two cases classed as idiopathic showed evidence of roughening of the articular surfaces of the vertebra, suggesting epiphysitis or osteochondritis.

Two cases presented three possible etiologic factors. A man, aged 26, gave a history of typhoid fever and meningitis preceding the deformity. This may have been anterior poliomyelitis, but the history of illness was not clear, and residual weakness or atrophy of the muscles was not present. The roentgenogram showed a suspicious asymmetry of the bodies of the fourth and fifth lumbar vertebrae, which was considered to be due to rotation, but which might have been a congenital anomaly. The case was classed as idiopathic.

The second case, that of a girl, aged 11, was also classed as idiopathic. The roentgenogram, however, showed large bilateral irregularly formed transverse processes of the fifth lumbar vertebra, and a spina bifida of the first sacral segment. There was also slight roughtening of the epiphyses of the lower dorsal vertebra, suggesting an epiphysitis.

Each of the larger groups of cases will be discussed separately.

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4. Scudder, Charles L.: A Determination of the Muscular Strength of Growing Girls and Its Relation to the Etiology, Treatment and Prognosis of Lateral Curvature of the Spine, Tr. Am. Orthop. A. **3**:84, 1890.

5. Lee, Benjamin: The Nervous and Muscular Element in the Causation of Idiopathic Lateral Curvature, Tr. Am. Orthop. A. **3**:80, 1890.

6. Adams, Z. B.: The Relation of Bony Anomalies of the Lumbar and Sacral Spine to the Causes and Treatment of Scoliosis, J. Orthop. Surg. **12**:45, 1914.

TYPES OF SCOLIOSIS

Idiopathic Scoliosis.—The static or idiopathic group was by far the largest, comprising seventy-nine cases, or 61 per cent of the total number. There was also a greater relative frequency in young girls in this group. While approximately 70 per cent of the total series of 125 cases occurred in females, sixty-three of the seventy-nine cases in the idiopathic group, or about 80 per cent, were females. The average age at which the deformity was noticed was 13 years, and the average age at the time of the examination was 15 years. In all cases the chief symptom was the deformity.

The diagnosis of idiopathic scoliosis can be made only by exclusion, as there is nothing characteristic either in the type or in the severity of the spinal deformity. The scoliosis varied from mild to extreme curvatures in all regions of the spine, both total and S-shaped curves being found. The typical deformity seen in adolescent girls is, however, a moderately advanced curve in the dorsolumbar region, with compensatory curves above or below.

As a rule, this group responds more favorably to treatment than the other groups, the curvature being easier to correct and to maintain corrected.

Paralytic Scoliosis.—Twenty-five cases were caused by anterior poliomyelitis. Of this group, sixteen occurred in females and nine in males. The average age at which the patient was examined was 15 years. Practically all gave a definite history of onset with paralysis, and the examination revealed muscular weakness, atrophy and the characteristic deformities of the extremities. In a previous study of anterior poliomyelitis, I [7] found scoliosis in 30 per cent of old cases. The deformity in paralytic scoliosis is often extreme, and is usually a total curve or one large curve and a small compensatory curve. Pain is a frequent symptom, being caused by impingement of the ribs on one another or on the pelvis. The deformity in advanced cases is difficult to correct or to maintain in even the slightest correction.

Congenital Scoliosis.—There are ten cases of congenital scoliosis in the series. As previously mentioned, only the gross malformations are included, other cases which show congenital anomalies being classed, in accordance with the predominating etiologic factor. All cases showing anomalous conditions of development, however, are charted in the table, and will be included in this discussion. As shown in the table, thirty-three cases, or 26 per cent of the total number, have developmental anomalies. Adams stated that on routine roentgen-ray examination, anomalies of the spine are found in about fifteen, or 16 per cent of

7. Mitchell, Joseph I.: The Residual Paralysis and Deformity of Anterior Poliomyelitis, J. Bone & Joint Surg. **7**:619, 1925.

all people. Since the percentage in this series is nearly double that found during routine examination, the congenital element must play an important rôle in scoliosis.

Of the various anomalies found, spina bifida occulta of the first sacral segment occurred most frequently, thirteen times, or 9.6 per cent. According to Sutherland,[8] spina bifida is found during routine examination in 5 per cent; therefore, the percentage of cases of scoliosis in which spina bifida is present is likewise nearly twice as large as the number found during routine examination.

In six cases there were six normally formed lumbar vertebra. In six cases there was asymmetry of the lumbosacral joint, and in three cases the transverse processes of the last lumbar vertebra were enlarged and of irregular shape. Wedging of the bodies of the vertebrae was noted in six cases. Cervical ribs were present in five cases; unilateral in

*Combination of Congenital Spinal Anomalies with Other Etiologic Factors**

	Spina Bifida	Lumbosacral Asymmetry	Irregular Transverse Processes 5th Lumbar Vertebra	Cervical Ribs	Wedge-Shaped Bodies	6th Lumbar Vertebra	Numerical Variations of Ribs
Idiopathic.....	8	3, **1** = 4	1, **2** = 3	1, **2** = 3	2, **3** = 5	4, **1** = 5	
Anterior poliomyelitis.....	3	..	..	..	..	1	
Empyema.....	1						
Congenital.....	1	2	..	2	..	..	6

* The numbers in bold faced type represent cases with more than one spinal anomaly present; total 33 cases; 44 congenital anomalies.

two and bilateral in three. The anomaly was considered as contributing to the scoliosis only in the unilateral cases.

Ten cases were undoubtedly caused by congenital malformation of the axial skeleton. Of these, four were in males and six in females. In five the deformity was noticed at or soon after birth, and in five it was not discovered until later in life. The average age at which examination was made was 13 years. A brief description of the congenital cases follows.

CASE 1.—A man, aged 24, had a mild right dorsal curve which had been present since birth. Roentgen-ray examination showed a large cervical rib on the left side.

CASE 2.—A boy, aged 7, had a severe right total scoliosis from the lower cervical region to the sacrum. The roentgenogram showed that there were ten ribs on the right and twelve ribs on the left side. There was synostosis between the three lower ribs on the left. This patient also had a supernumerary thumb on the right hand.

8. Sutherland, Charles G.: A Roentgenographic Study of Developmental Anomalies of the Spine, J. Radiol. **8**:357, 1922.

CASE 3.—A girl, aged 17, presented a severe right dorsal curve. There were thirteen fully developed ribs on the left and fourteen ribs on the right side. There was considerable rotation, with distortion and wedging of the vertebra from the third to the seventh dorsal. There was also a spina bifida of the fifth lumbar vertebra with absence of lamina and articular process on the right.

CASE 4.—A youth, aged 19, showed a moderate dorsal scoliosis convex to the right. The fifth and sixth dorsal vertebrae were wedge-shaped, being narrower on the left side. The sixth rib on the right was widely separated from the fifth and seventh; and there was a partially developed rib between it and the fifth rib, synostosed to the sixth. There were thirteen ribs on each side.

CASE 5.—A girl, aged 16, complained of a very short neck and a mild dorsal kyphoscoliosis, convex to the right. The roentgenogram showed a congenital anomaly of the lower cervical spine. One half of the body of the seventh cervical vertebra was absent and a well developed rib was articulated on the left.

CASE 6.—A youth, aged 19, was examined for an S-shaped curve convex to the right in the cervicodorsal region, convex to the left in the upper dorsal, and convex to the right in the middorsal region. On roentgen-ray examination, thirteen ribs were demonstrated on the right side, the last one being rudimentary. The left half of the first dorsal vertebra and the first rib were absent. The left ninth rib was absent, thus making eleven ribs on the left. The lumbosacral joint was asymmetrical. Figure 1 shows the roentgenogram of the spine.

CASE 7.—A girl, aged 1 year and 3 months, had a right dorsal curve at birth. The roentgenogram revealed absence of the left half of the seventh and eighth dorsal vertebrae and of the seventh and eighth ribs. The sixth and ninth vertebrae were wedge-shaped.

CASE 8.—A girl, aged 13, presented a dorsolumbar scoliosis convex to the right. There was a congenital malformation of the fifth lumbar vertebra and the lumbosacral articulation.

CASE 9.—A boy, aged 10, had a right lumbar curve with anomalous development of the lumbosacral joint.

CASE 10.—A boy, aged 10, presented a right dorsal scoliosis. The left leg was shorter than the right, and the left foot was clubbed. The eighth, ninth, tenth and eleventh dorsal vertebrae were wedge-shaped, being narrower on the left and anteriorly than on the right and posteriorly. There were thirteen fully developed ribs on each side and a small fourteenth rib on the left. This extra rib articulated with the first lumbar vertebra.

Empyema.—Four cases were caused by empyema, three in boys and one in a girl. There was a history of pneumonia and empyema in early childhood, followed by collapse of the lung. The deformity in this group may be great and is resistant to treatment because the collapsed lung cannot be made to expand the wall of the chest.

Associated Conditions.—Various associated conditions were noted, some of which were dependent on the same etiologic factor as the scoliosis, and none of which should be considered as etiologic factors in themselves, because the relative frequency of each is small in comparison with the total number of cases.

The first to be considered is inequality in length of the legs. In only ten cases, excluding the paralytic group, was there a difference

in the length of the legs. This inequality varied from ¼ to ¾ of an inch. Scoliosis does not, as a rule, follow inequality in the length of the legs. It is not uncommon to find an otherwise normal person with shortening on one side. On the other hand, in many cases of extreme scoliosis there is no inequality. In many cases with extreme inequality,

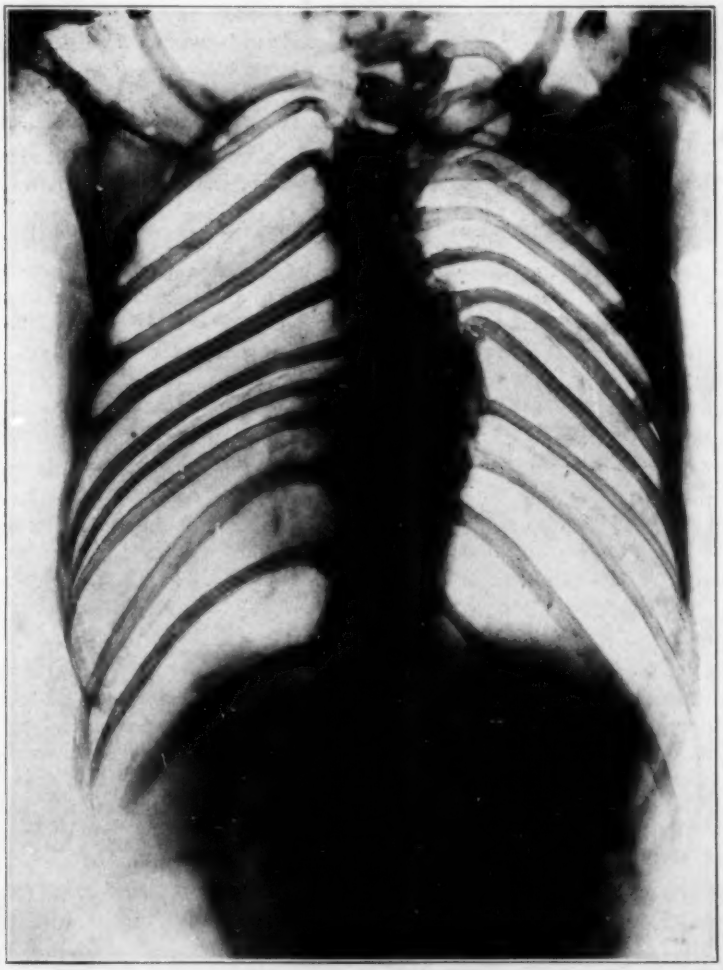

Fig. 1 (case 6).—Roentgenogram showing congenital scoliosis. Note the absence of the left half of the first dorsal vertebra, and the first and ninth ribs.

as in infantile paralysis, there is no fixed deformity of the spine. There is a tilting of the pelvis when the patient stands and a compensatory lateral curve of the spine which disappears, however, on sitting or lying.

Flatfoot, of a degree severe enough to demand treatment, was observed only six times in the nonparalytic cases. Kyphosis, round

shoulders and increased lumbar lordosis were observed in a small percentage of cases. Four cases in the series showed evidence on roentgen-ray examination of a disturbance of the epiphyseal areas of the vertebrae, suggesting an osteochondritis or epiphysitis. As mentioned previously, there was muscular weakness, atrophy and deformities in the paralytic group; and skeletal malformations in the congenital cases. One case showed torticollis, one a supernumerary thumb, and one a talipes equino varus of congenital origin. Twenty-two cases showed albuminuria.

TREATMENT

With the introduction of ankylosing operations on the spine, interest in the treatment of scoliosis has been greatly stimulated. Fusion of the affected region of the spine offers a practical and efficient method of preventing the increase of the deformity, and when combined with thorough preoperative correction gives a more definite goal toward which both patient and surgeon may strive, than can be anticipated in the majority of cases in which other methods of treatment are used.

As previously mentioned, our study of treatment is confined to thirty-six cases. These patients are unselected, except for the fact that they have cooperated sufficiently well in treatment and have returned for observation to enable us to determine something of the effect of treatment and the permanency of the results. Ten of these cases have been fused.

Corrective Treatment.—We have endeavored to adapt the treatment to the individual requirements of the various patients. In some cases, two or more methods have been employed, and in many cases, several casts have been applied successively, before a satisfactory degree of correction has been secured. The standard of maximum correction must be determined not only from the external contour, but also by the roentgenogram. However, one must often be satisfied with a good cosmetic result. The duration of the corrective period usually varies from eight to twelve weeks, but in a few cases has been as long as eighteen months.

Traction on a convex stretcher frame, as advocated by Whitman,[9] was used on six patients, four of whom were later operated on. It has been found effective, especially in the paralytic cases, in extending the spine, lessening the angular deformity of the ribs and expanding the chest on the contracted side.

Most satisfactory results have been obtained in suitable cases with the turnbuckle cast described by Lovett and Brewster.[10] It was used

9. Whitman, Royal: Observations on the Operative Treatment of Scoliosis, J. Orthop. Surg. **3**:330, 1921.

10. Lovett, R. W., and Brewster, A. H.: The Treatment of Scoliosis by a Different Method from that Usually Employed, J. Bone and Joint Surg. **6**:847, 1924.

on thirty-three patients in this series, five of whom were later operated on. As designed by the originators, it is most efficient in those cases in which there is a single curve at the dorsolumbar region. In cases of double curves, it has been demonstrated, however, that by correcting the dorsolumbar curve, compensatory curves above and below may be lessened. The cast may be extended to the knee on the convex side for correction of the lumbar curves.

The cast from chin and occiput to pelvis to be applied under traction on a special frame, described by Hibbs,[11] is undoubtedly the most efficient method for correcting high dorsal curves, and in this series it has been

Figure 2

Figure 3

Fig. 2.—Correction of scoliosis by turnbuckle cast.

Fig. 3.—Correction of scoliosis in cast applied on Hibbs' traction frame. Note the canvas band for lateral traction.

reserved for that type of deformity. Eleven patients have been treated by this method, seven of whom have been fused.

Four patients were treated by fenestrated casts and pads, three of whom were operated on.

Since the upright position favors the progress of the deformity, we believe that recumbency is an important factor in treatment. This is of course essential to the Whitman method, but when practical, we

11. Hibbs, Russel A.: A Report of 59 Cases of Scoliosis Treated by the Fusion Operation, J. Bone & Joint Surg. **6**:3, 1924.

have insisted on continuous recumbency also for the patients being treated by the Hibb's or turnbuckle type of cast. When it was not possible to maintain continuous rest in bed, we have advised long rest periods daily.

Retention By Braces.—When the maximum degree of correction has been secured, the problem arises of how to maintain it. Postural exercises and braces have been employed in twenty-six cases in this series. In addition to the various standard spinal braces, the brace here described has proved efficient in maintaining the correction in the lower dorsal and lumbar regions. It is comfortable and is easily removable

Fig. 4.—Leather corset for scoliosis which has been made on corrected model.

for bathing and exercises. This brace has been built on the same principle as the turnbuckle cast. A plaster of paris model of the trunk is made and removed immediately. When thoroughly dry, it is cut transversely at the level of greatest deformity. The segments are then separated on the concave side similar to the turnbuckle cast. A few turns of plaster bandage hold the model in this corrected position. The form is then filled with plaster cream, and a torso of the patient is secured in the corrected position. Over this torso, a leather corset is made and is reinforced with steel strips. For the high dorsal curves, a head support must be attached to the brace.

Operative Treatment.—We believe that in many cases, operative fusion is the method of choice. The two classic methods of spinal

fusion, the Albee graft and the Hibbs' technic, are both unsuitable in scoliosis. The straight rigid Albee graft is difficult to adapt to the curve of the deformity. In Hibbs' operation, the laminae and articular faucets are difficult to reach on the concave side. It has been our experience that an osteoperiosteal graft from the tibia was the most satisfactory method. This may be combined with the Hibbs' operation and supplies a large mass of new bone which is flexible and easily accommodated to the area of deformity. The osteoperiosteal graft was used in eight of the ten cases in which operation was performed; in two cases with prominence of the ribs, a portion of the rib was resected and used as an additional graft in combination with the Hibbs' fusion.

As in all operations on the spine, fusion is often accompanied by some degree of postoperative shock. This can be best prevented by the preliminary adjustment of the patient in the prone position on sandbags, so that the respiration, already limited by the deformity, is not further impaired. Stimulants are frequently necessary and should be readily available. There have been no deaths in this series.

A brief summary of the cases in which operation was performed follows.

CASE 1.—*Paralytic scoliosis.* A youth, aged 19, was examined Sept. 30, 1921. He gave a history of infantile paralysis at 2 years of age. Examination showed an extreme right dorsal, left lumbar scoliosis. Fenestrated casts were applied to correct the deformity. On March 1, 1922, the spine was fused, the Hibbs' technic being used and a segment from the fourth rib as a graft. The result has been good. Fusion of the spine was solid.

CASE 2.—*Paralytic scoliosis with congenital anomaly.* A boy, aged 13, was first seen April 2, 1923. The deformity followed anterior poliomyelitis at 4 years of age and was progressing rapidly.

The examination showed a severe total curve convex to the right. There was marked rotation of the bodies of the dorsal vertebrae with an angular projection of the ribs on the right side posteriorly. The right side of the chest was narrowed, and the right shoulder was higher than the left. The roentgenogram showed in addition to the deformity, six lumbar vertebrae and a spina bifida of the first sacral segment.

Traction by the Whitman method on a curved Bradford frame was applied for eight weeks; further correction was secured by fenestrated casts. On August 22, the spine was fused from the sixth dorsal to the first lumbar vertebrae. In addition to Hibbs' technic, a 2 inch (5 cm.) section was removed subperiosteally from the prominent portion of the sixth, seventh and eighth ribs. This bone was fragmented and placed in the graft bed. The wound healed promptly, and eight weeks later, the boy was up, wearing a spinal brace.

The result has been excllent. When observed in June, 1927, the fused area of the spine was solid. The contour of the spine was much improved, and there has not been any increase of the deformity.

CASE 3.—*Paralytic scoliosis.* A girl, aged 19, was examined May 22, 1925. She had had an anterior poliomyelitis at the age of 10, and the deformity of the spine had been noticed a few months later. She had worn plaster of paris jackets for two years, but in spite of this the deformity was progressing, and she

Fig. 5 (case 3).—Before treatment.

Fig. 6 (case 3).—After correction and fusion of spine.

complained of constant pain in the lower part of the spine, referred about the right iliac crest. The spine showed a marked total scoliosis convex to the right. The right leg was one half inch (1.27 cm.) shorter than the left, and both legs showed muscular atrophy and weakness.

On July 29, after six weeks' preliminary traction on a curved Bradford frame, a Hibbs' fusion operation was performed from the seventh dorsal to the second lumbar vertebra. This operation checked the progress of the deformity, but pain persisted. On Dec. 1, 1926, she returned for a second operation, at which time the spine was fused from the second lumbar vertebra to the sacrum, an osteoperiosteal graft from the tibia being used.

Figure 7

Figure 8

Fig. 7 (case 5).—Before treatment.

Fig. 8 (case 5).—After correction of deformity of legs and fusion of spine.

Roentgenograms made on April 4, 1927, showed solid fusion of the spine from the eighth dorsal vertebra to the sacrum. The contour of the back was good and the pain was much less, though she still had an occasional pain on the right side.

Case 4.—*Destructive lesion.* A girl, aged 10, was first examined on June 16, 1921, for kyphoscoliosis. The child complained of pain in the back and tired easily. Roentgen-ray examination of the spine showed wedging of the body of the twelfth dorsal vertebra, fusion of the bodies of the first and second lumbar vertebrae and irregularity in outline of the bodies of the remaining lumbar vertebrae. A plaster of paris cast was applied, but treatment was soon discontinued.

On May 21, 1925, she returned for observation. The deformity had increased, and the pain was more severe. Examination showed a right dorsal left lumbar curve with a rounded kyphos in the lumbar spine. The roentgenograms were similar to those previously made and did not show any increase in destruction in the vertebral bodies. The impression was that the condition resulted from a low

grade inflammatory lesion, an osteochondritis or an epiphysitis of the spine. Some correction was secured by the plaster cast, and on August 20, a fusion operation was performed from the tenth dorsal to the third lumbar vertebra, an osteoperiosteal graft being used.

Convalescence was uneventful and the result excellent. When examined on June 2, 1927, there had been complete relief from pain, no increase in deformity, and the fused area of the spine was solid.

CASE 5.—*Paralytic scoliosis.* A girl, aged 15, was examined Aug. 28, 1923. She had had an attack of anterior poliomyelitis at 8 years of age, followed by extensive paralysis and contractures of the trunk and both lower extremities. There was a total left scoliosis from the second dorsal vertebra to the sacrum.

Figure 9

Figure 10

Fig. 9 (case 6).—After correction of deformities of legs and before treatment of spine.

Fig. 10 (case 6).—After correction and fusion of spine.

The deformities of the legs were first corrected. On June 8, 1925, a turnbuckle cast was applied and changed at intervals for three months. On Sept. 7, 1925, the spine was fused from the seventh dorsal to the second lumbar vertebrae, an osteoperiosteal graft being used. Two weeks later, when the wound was healed, a turnbuckle cast was reapplied to maintain the correction during the time of consolidation of the fused spine. A spinal brace was fitted on Dec. 28, 1925. The result was good. When observed May 8, 1926, the spine was holding in improved position and the patient was walking better with crutches.

CASE 6.—*Paralytic scoliosis.* A girl, aged 14, was examined Sept. 23, 1923. At 3 years of age, she had had anterior poliomyelitis with residual deformity of the spine and both legs. The spine showed a total curve to the left. The deformity of the feet and legs was corrected. Correction of the spinal deformity

was begun on Nov. 17, 1925, by traction on a curved stretcher frame. Three months later, a turnbuckle cast was applied. On July 8, 1926, the spine was fused from the tenth dorsal to the second lumbar vertebrae, an osteoperiosteal graft being used. Three weeks later, a cast was applied on a Hibbs' traction frame. A leather jacket was fitted on Oct. 9, 1926, and the patient was allowed out of bed.

The result was excellent. When observed on April 11, 1927, the contour of the spine was good and fusion solid.

CASE 7.—*Idiopathic scoliosis, with congenital anomaly.* A woman, aged 25, married, was examined on Aug. 15, 1926. The deformity was first noticed when the patient was 17 and had become exaggerated during each of two pregnancies. Pain in the back had been constant for two years. Examination showed a left dorsal, right lumbar curve. The roentgenogram showed wedging of the eighth,

Figure 11

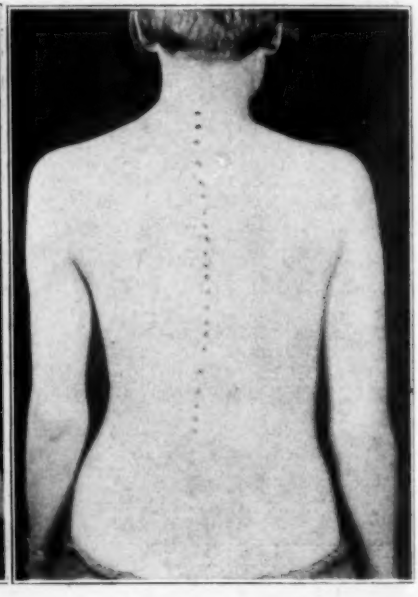

Figure 12

Fig. 11 (case 8).—Before treatment.
Fig. 12 (case 8).—After correction and fusion of spine.

ninth and tenth dorsal vertebrae, the left side of the bodies being narrower. There was also a spina bifida of the first sacral segment. A turnbuckle cast was applied and worn for seven weeks. On October 14, an osteoperiosteal graft was employed to fuse the spine from the sixth dorsal to the twelfth dorsal vertebrae. Two weeks later, a plaster cast was applied.

When observed on June 25, 1927, the position of the spine was much improved. Fusion was solid, and there had been complete relief from pain.

CASE 8.—*Idiopathic scoliosis.* A girl, aged 16, was examined on Sept. 18, 1926. The deformity was first noticed by a dressmaker one year previously. The girl complained of tiring easily and pain in the back and abdomen. Examination showed a moderately severe curve, right dorsal, left lumbar. A cast was applied

from chin and occiput to pelvis on the Hibbs' frame, and the deformity corrected as shown by roentgen-ray examination. On October 16, the spine was fused from the fourth to the tenth dorsal vertebrae, an osteoperiosteal graft being used. When the wound was healed, a second Hibbs' cast was applied to maintain the correction.

The result was excellent. The spine was almost straight, and in June, 1927, the patient was allowed to discontinue wearing the brace.

CASE 9.—*Paralytic scoliosis.* A girl, aged 16, was examined May 28, 1924. She gave a history of infantile paralysis at 4 years of age, followed by paralysis of the trunk and lower extremities.

After operation on the legs, she was able to walk with braces and crutches. In June, 1926, she was placed on a convex stretcher frame with traction to correct

Figure 13

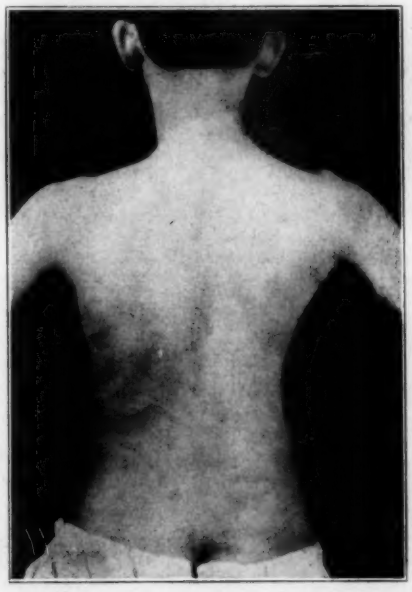

Figure 14

Fig. 13 (case 10).—Before treatment.
Fig. 14 (case 10).—After correction and fusion of spine.

the right total scoliosis. In August, 1926, a turnbuckle cast was applied, and on Oct. 19, 1926, the spine was fused from the eighth dorsal to the second lumbar vertebrae, an osteoperiosteal graft being used. Two weeks later, a cast was applied.

The result was excellent.

CASE 10.—*Paralytic scoliosis.* A boy, aged 15, was examined Nov. 19, 1924. The history of onset was indefinite; the deformity of the spine and the slight, but definite weakness and atrophy of the lower extremities was pathognomonic of anterior poliomyelitis. There was a severe grade of scoliosis, left dorsolumbar and right lumbar. A spinal brace was worn for a time, but the deformity increased. Following a series of casts applied on the Hibbs' traction frame, a

spinal fusion was performed on Jan. 14, 1927. An osteoperiosteal graft was placed from the fifth dorsal to the first lumbar vertebrae.

Two weeks after operation, another cast was applied, and later a spinal brace.

The contour of the spine was much improved, and the progress of the deformity was checked.

CONCLUSIONS

1. The etiology, in the majority of cases of scoliosis, is still obscure. In a study of 125 cases, 61 per cent were of the idiopathic type.

2. The treatment, if successful, must be adapted to the individual case.

3. Fusion of the affected region of the spine offers a practical and efficient method of preventing the increase of the deformity. It is advised in all paralytic cases and in other cases in which progress of the deformity is anticipated.

4. Careful preoperative correction is essential.

5. The use of the osteoperiosteal graft, in fusion, is most satisfactory.

BRONCHOBILIARY FISTULA

REVIEW OF THE RECORDED CASES OTHER THAN THOSE DUE TO ECHINOCOCCUS AND AMEBIC ABSCESS *

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One of the most dramatic pictures in medical practice is that of a patient torn by racking bouts of coughing accompanied by the expectoration of a bright yellow or brilliant green sputum. The abject misery of these sufferers as well as the relative infrequency of the condition at once commands interest.

A recent instance led us to consult a number of surgeons of experience; all of them had very little acquaintance with this lesion, which indicates how seldom it is seen in the practice of any one physician.

DEFINITION

In the literature a number of cases are reported which closely simulate bronchobiliary fistulas, but which really are not instances of this condition. Some of these patients had hepatic abscesses which ruptured through the diaphragm into the pleura. These cases should be considered properly as hepatopleural or pleurobiliary fistulas. In other cases, authors reported that an abscess had emptied itself through the lung and the bronchi, but bile had never been noted in the expectoration. In still other types, a rupture had taken place through the bronchi followed by complete healing or death in a few days; perhaps traces of bile had been present in the sputum on one examination. This class includes many cases of ruptured tropical abscess of the liver. None of the foregoing conditions will be considered as examples of the true bronchobiliary fistula. We define a bronchobiliary fistula as a communication between the biliary tract and the bronchial tree by which bile is emptied through the sputum over a considerable period of time. The essential factors for the maintenance of a biliary fistula between the liver and the bronchi seem to be a cavity either in the substance of the liver or walled off in the subphrenic space; or dilated bile ducts which are prevented from emptying in their normal manner below by an obstruction of some kind. The increasing pressure within the cavity or ducts keeps the fistula open until it becomes a well established channel.

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HISTORICAL

No attempt was made to collect a group of these cases for study until 1890. Courvoisier [1] at that time reviewed twenty-four cases of hepatopleural and hepatobronchial fistulas reported in the literature. Six of these patients were observed only during life, but the clinical picture was beyond mistake. The other eighteen fistulas were studied at postmortem examination as well. Cayley's [2] patient had only a hepatopleural fistula. Legg's [3] patient had a hepatopleural and a pericardial fistula. In Pasturaud's [4] case, a subphrenic abscess ruptured into the bronchi, but mention is not made of bile in the sputum, and postmortem examination showed that the liver tissue was not involved; in Riedel's [5] case, an abscess of the liver perforated the abdominal wall, the pleural cavity and the transverse colon, but a pulmonary fistula was not found. These cases do not fit our definition and will consequently be omitted. In 1897, Graham [6] added two of his own reports to the literature, and summarized an additional nine which had been published since Courvoisier's work. Ido and Yasuda [7] made a comprehensive review of the literature up to 1912, and succeeded in compiling reports of forty-eight cases of biliary pulmonary and pleural fistulas in addition to their own. In 1923, Oliani [8] brought the number of these cases reported up to sixty-three. His excellent paper included discussions of biliary pulmonary fistulas due to every cause, over 40 per cent being of echinococcus origin. The case summaries of the latter class of fistula and those due to amebic abscess have been omitted from this review, as they concern more properly tropical or subtropical regions. Biliary pulmonary fistulas due to echinococcus disease are numerous, probably out-numbering all other causes combined. Sendler [9] found a total of sixty-eight cases due to this cause. Smitten [10] found seventy-one cases of hepatopleural and bronchobiliary fistulas, only four being reported from his native country (Russia). He added two of his own cases to this series. In sixty-one of these seventy-three, the sinus led into the bronchi and in twelve into the pleural cavity. A majority

1. Courvoisier, L. G.: Casuistisch-Statistische, Beitr. z. Path. u. Chir. der Gallenwege, 1890, p. 111.
2. Cayley, W.: Tr. Path. Soc., London **17**:160, 1866.
3. Legg, J. W.: Tr. Path. Soc., London **25**:133, 1874.
4. Pasturaud: Bull. Soc. anat. de Paris **49**:189, 1874.
5. Riedel: St. Peters. med. Wchnschr., 1885, p. 157.
6. Graham, J. E.: Brit. M. J. **1**:1397, 1897.
7. Ido, Y., and Yasuda, S.: Beitr. z. path. Anat. **52**:577, 1912.
8. Oliani, E.: Ann. ital. di. chir. **2**:1075 and 1288, 1923.
9. Sendler, O.: Ein Fall von Leberbronchusfistelbildung durch Echinokokkus enstanden und durch Choledochussteine Kompliziert, Jena, A. Kampfe, 1910, p. 45.
10. Smitten, A. G.: Russk. Klin. **7**:252, 1927.

of these fistulas would not meet the requirements of our more restricted definition of a true biliarybronchial fistula.

Ido and Yasuda regarded Heaton's [11] (1869) as the oldest report in the literature. This mistake could not have occured if these writers had consulted Courvoisier's report, which includes the cases of Mandard [12] (1854), Lebert [13] (1857) and Bristowe [14] (1858)—all bronchobiliary fistulas which antedated Heaton's case. Peacock [15] (1850) gave a clearcut picture of two patients who suffered with this type of fistula following echinococcus disease of the liver; similar cases were reported also by Robin and Mercier [16] (1855); Cadet de Gassicourt [17] (1856) and Tierfelder [18] (1860). Foard [19] (1866) gave a typical example, though doubtless the journal in which he published the report was not available. The thesis of Rapp [20] (1867) (which could not be obtained) also has the suggestive title "Ein Leber Lungenfistel."

REPORT OF A CASE

History.—Mrs. A. M. R., aged 50, was admitted to the Strong Memorial Hospital on May 3, 1926, because of a violent, paroxysmal cough. She had had measles and whooping cough as a child and typhoid fever at 17, but with these exceptions, her health had been excellent up to four or five years before admission. At that time, she had had attacks of typical gallstone colic, which came on every three or four months. These attacks consisted in severe pain along the right costal margin, accompanied by vomiting, but never by jaundice. One year before admission, an infected gallbladder filled with stones was removed at another hospital. A week after operation, fever and pain in the region of the wound occurred, and by the aid of roentgenograms a diagnosis of subphrenic abscess was made. The wound was reopened and explored, but an abscess was not found. The fever continued, and a cough developed with expectoration of yellow sputum. The patient stayed in the hospital in this condition for three months, and then slowly improved on going home, till she was almost well eight months after operation. About six weeks before admission (March, 1926), she had an attack of vomiting that lasted a day, followed by the recurrence of fever, cough and yellowish sputum. The coughing occurred in bouts lasting about two hours, closely resembling whooping cough. These attacks left the patient so weakened that she remained in bed. She raised small amounts of thin, mucoid sputum with each fit of coughing, but not a large quantity. The sputum was yellow, grass green or white, and tasted "bitter as gall." There was some pain in the

11. Heaton, J. D.: Brit. M. J. **2**:8, 1869.
12. Mandard, L. E.: Thèse de Paris, no. 301, 1854, p. 17-18.
13. Lebert, quoted by Oliani: Traité d'anat. path. gen. et spec. et atlas, Paris, 1857.
14. Bristowe, J. S.: Tr. Path. Soc., London **9**:285, 1858.
15. Peacock, T. B.: Edinburgh M. J. **74**:33, 1850.
16. Robin and Mercier: Mém. Soc. biol., 1855, p. 116.
17. Cadet de Gassicourt: Thèse de Paris, 1856.
18. Tierfelder, quoted by Sendler: Rostok, 1860.
19. Foard, A. J.: New Orleans M. & S. J. **19**:192, 1866.
20. Rapp, A.: Ein Leber Lungenfistel, Würzburg, 1867.

chest, but no gastro-intestinal symptoms. She was most comfortable in a half sitting position, which reduced her cough to a minimum.

Physical Examination.—The patient had evidently been a well developed and healthy woman, but she now appeared tired, drawn, and pallid from the effects of the violent, racking cough which came on at frequent intervals. Nothing abnormal was found except for the chest and upper part of the abdomen. The chest was symmetrical, expansion being equal on both sides, and there was tenderness on pressure over the margins of the right ribs. An area, slightly dull on percussion, was made out in the paravertebral region near the base of the right lung. The breath sounds were bronchovesicular at the right apex posteriorly; and from the right interscapular region to the base and outward to the posterior axillary line, there was rather loud bronchial breathing. There was increased vocal and tactile fremitus in this area, and a few medium râles were heard. The anterior parts of the lungs and the left side were clear. There was a linear scar in the right upper quadrant of the abdomen with a small drainage scar adjacent. When the patient coughed or sat up, there was herniation of the wound. The fingers showed linear markings and incurving of some of the nails, but no clubbing of the finger tips.

The temperature on admission was 37 C. (98.6 F.); pulse rate, 84; respiration, 22 and blood pressure, 104 systolic and 66 diastolic. The red blood count was 3,568,000 and the hemoglobin reading, 82 per cent (Sahli). The blood smear did not show any abnormalities. The blood count revealed 7,700 white blood cells; polymorphonuclears, 76 per cent; lymphocytes, 20 per cent and mononuclears, 4 per cent. The urine was clear with an acid reaction: the specific gravity was 1,020 and albumin and sugar were not present. Microscopically, the urine was normal.

May 4, 1926: About 75 cc. of dark yellow, mucoid, odorless sputum was expectorated the day before. A Gram stain showed a few mixed bacilli. An acid-fast stain did not reveal any tubercle bacilli. The mercuric chloride test was positive for bile.

May 5: Sixty cubic centimeters of yellow sputum was expectorated. The urine contained a slight trace of bile.

May 6: One hundred cubic centimeters of yellow sputum was expectorated that gave a strong nitric acid test for bile. The stool was large and light colored, but gave a positive reaction for bile. The Wassermann test on the blood was negative. Plasma bilirubin was slightly positive in a 1:2 dilution. The results of the phenolsulphonphthalein test for kidney function are given in the following table:

Phenolsulphonphthalein Test for Kidney Function

Hours	Total Output, Cc.	Phenolsulphonphthalein Percentage
1	460	20
2	100	12
Total	560	32

Roentgen-Ray Examination.—The roentgen-ray examination performed by Dr. S. L. Warren on May 3 and 4 showed that the excursion of the left side of the diaphragm was increased, and the costophrenic angle was clear and sharp. The left lung transmitted the rays more brilliantly than usual in the apex and upper lobe so that the larger bronchi were easily visible. The region of the left hilum had increased considerably in density, and a marked amount of peri-

bronchial thickening extended from the hilum toward the apex. There was considerable mottling around the bronchi at the left base, suggesting a localized area of bronchopneumonia. The heart was pulled toward the right and fixed in position. The right side of the chest presented marked peribronchial thickening easily traceable out toward the apex and the periphery. The masses in the hilum extended far out and obscured the right border of the heart. The right side of the diaphragm did not move with respiration, and its outline was obscured by a uniformly dense shadow, more dense than the shadow of the liver, which came to the level of the ninth rib in the posterior axillary line. The outline of the liver was low, the upper border coming down almost to the level of the eleventh rib. Considerable haziness and mottling of the base of the right lung served to confuse the picture further. A tract about 2 cm. wide extended from the hilum along the course of the right lower main bronchus into the dark mass already described. When the patient coughed a column of fluid could be seen to shoot upward with great rapidity from the region of the right side of the diaphragm along this tract and into the trachea, and the patient expectorated bile-stained sputum. The column was seen to be projected to the level of the clavicles and was uniform in diameter for the whole distance. In the lateral view, the posterior mediastinum was obliterated, except in the supracardiac area just below the clavicles. The mass in the region of the right side of the diaphragm seemed to be slightly posterior to the middle, but its greatest diameter occupied the region normally below the dome of the diaphragm, midway between the front and the back and about 6 cm. from the midaxillary line. The lung apparently was well expanded around the upper part of the abscess, especially laterally and posteriorly. The merging of the abscess and the shadows of the liver made accurate localization of the abscess cavity impossible.

May 10: Operation was performed. About 4 inches (10.16 cm.) of the eleventh rib was resected posteriorly under procaine hydrochloride anesthesia. The lung was not adherent to the pleura, so that pleural reflection was identified, dissected free and pushed upward without opening the pleural cavity. The diaphragm was sutured to the intercostal muscles in two layers over this transplanted pleural space. The diaphragm was then divided and the subphrenic space explored. It was necessary to give the patient nitrous oxide and oxygen during this manipulation. The entire upper surface of the liver posteriorly was firmly attached to the under surface of the diaphragm by adhesions which were gradually broken up until the whole hand could be introduced over the dome of the liver. The surface of the liver felt roughened and nodular. After the adhesions between the liver and the diaphragm over a large area had been broken through without encountering a subphrenic abscess, it was considered wise to terminate the operation at this point as it was hoped that by repeating the roentgen-ray study with a drainage tube in the subphrenic area, more accurate localization would be possible. Accordingly, two Penrose drains and a large sized rubber tube wrapped with iodoform gauze were inserted and sutured in place. The operation was well borne.

For the next four days, the patient's condition was decidedly poor. The pulse was feeble and rapid; the blood pressure was low; and the patient suffered from nausea and vomiting, violent attacks of coughing and a moderate abdominal distention. She could not retain fluids by mouth, so that it was necessary to use the rectal, intravenous and subcutaneous routes. It became apparent that unless the abscess were evacuated soon, the patient would die from exhaustion. A bedside roentgenogram taken immediately after operation showed the rubber tube to be 1 cm. lateral to and below the abscess cavity. The blood chemistry was

studied on May 14 and it was found that the total amount of blood chlorides was 0.469 mg. per hundred cubic centimeters; the carbon dioxide combining power, 54.5 per cent; and the nonprotein nitrogen, 26.6 mg. per hundred cubic centimeters.

The second operation, performed under nitrous oxide, consisted in removing the drains, introducing the hand over the dome of the liver, breaking through into an abscess cavity which discharged a large amount of foul smelling colon bacillus pus and reintroducing three drains.

The prognosis appeared grave, but the operation itself caused little disturbance in the patient's condition. She was given physiologic sodium chloride solution by hypodermoclysis and a transfusion of blood following the operation.

Cultures of the pus obtained at operation grew *B. coli-communis* and *Staphylococcus aureus.*

Improvement began with the cessation of vomiting and continued steadily with a free drainage from the wound and a gradual diminution in the cough and the amount of sputum. The sputum still showed a trace of bile by Heller's nitric acid test one week after the operation, and *Bacillus coli* was grown from the sputum on the same date. There was a sudden change for the worse on May 22 with a rise in temperature to 40 C. (104 F.); the pulse rate went up to 140 and the respiration to 40. There was a chill, marked dyspnea and cyanosis, restlessness, a cold clammy perspiration and a drop in blood pressure to 72 systolic and 49 diastolic. The percussion note over the right side of the chest was tympanitic; the breath sounds were absent, there was little movement of the right side of the thorax. The heart was displaced far toward the left axilla, and the breath sounds on the right were exaggerated. The white blood count was 23,000. It was evident that there was a pneumothorax; this was confirmed by a bedside roentgenogram.

Aspiration of about 800 cc. of air from the right pleural cavity resulted in an almost miraculous change in the general condition and immediate relief from all distressing symptoms. We do not understand the mechanism of this pneumothorax. If the perforation occurred beside the fistula through the diaphragm, it is difficult to understand why infection of the pleural space did not occur. Wherever the perforation was, it was sealed off immediately, as the pneumothorax did not recur following one aspiration of air. From this time on, the patient improved rapidly, so that the drainage had practically ceased by May 30, and the cough was almost gone A furuncle on the right thigh was the only other complication during convalescence.

On June 19, she was discharged from the hospital with a small area of granulations at the site of the incision: there was no pain or evidence of inflammation in the region. An occasional dry cough occurred without sputum, and the patient seemed to be in fairly good general condition. The thorax looked symmetrical; the respiratory movements were good. The percussion note was resonant, posteriorly and anteriorly, but impaired at the base of the right side of the thorax, both laterally and in the back. The right base was percussed out from about 2 to 3 cm. higher than the left, and the lung did not descend as much here as on the left. The breath sounds were distant with a slight bronchovesicular quality, but râles were not heard. The edge of the liver was not painful on palpation.

On July 9, 1926, the patient reported for examination with her wound closed and without complaints except that rarely, she had a dry cough, never accompanied by sputum; and residual weakness resulting from her long confinement in

bed. She was recovering her strength rapidly and was about to go on a trip to the country to complete her convalescence.

On Oct. 15, 1926, she visited the clinic, having regained her normal health.

Summary.—A patient with cholelithiasis and cholecystitis was treated by cholecystectomy. Removal of the gallbladder was followed during the immediate postoperative period by what was diagnosed as a subphrenic abscess. An exploration through the original anterior abdominal incision did not reach the abscess, and it gradually worked its way through the diaphragm and into the lung, which had become adherent to the diaphragm, and thence into a bronchus. There was no obstruction to the normal discharge of bile, as was demonstrated by the presence of bile in the stools prior to operation, and by the subsequent course of the patient when the discharge of bile to the surface was stopped; there was no evidence of biliary obstruction. We assume that the subphrenic abscess communicated with intrahepatic biliary ducts through an abscess cavity in the sub-

Fig. 1 (Morton and Phillip's case).—Subphrenic abscess at the dome of the diaphragm which caused a bronchobiliary fistula by breaking through.

stance of the liver. Drainage of this infection with its complicating leakage of bile through the bronchial tree was not sufficient to cause cure. With wide open external drainage, healing and obliteration of the subphrenic and intrahepatic infected cavities was possible. The fistulous tract to the surface, through the diaphragm, lung and bronchial tree closed as fistulas usually do when there is no longer material to be drained through them.

ETIOLOGY

Bronchobiliary fistula without doubt is caused more often by echinococcus disease than by any other single agency. Sendler [9] found sixty-eight cases of fistulas due to this cause. Many instances of amebic abscess have also been recorded. Miqiniac said that 17 per cent of tropical abscesses perforate into a bronchus (Smitten).[10]

Besides the foregoing conditions, we have been able to find in the literature reports of only forty-nine really authentic bronchobiliary fistulas that fit our definition.

The majority of these fistulas were caused by suppurative processes affecting the subphrenic space, liver, gallbladder and biliary passages. Suppuration followed biliary tract disease, cholelithiasis or cholecystitis —in at least twenty-two instances, verified by operation or autopsy— and probably it was the primary cause in thirteen other cases not proved. These patients either recovered spontaneously or postmortem examination was not performed. In some cases, more than one cause operated with the suppurative process to produce the fistula. Eschenhagen [21] reported such a case in which there was a stone in the common duct and a tuberculous process which spread along the fistula from a tuberculous cavity in the apex of the right lung. Ido and Yasuda [7] and MacDonald [22] reported cases of biliary tract disease and syphilis as possible etiologic factors. In MacDonald's case, antisyphilitic treatment caused the bile to flow in the normal manner, and thus closed the bronchial fistula. This was probably due to the resolution of a gumma which was blocking the common duct. The diagnosis of biliary tract disease was made only when the patient coughed up some calculi at a later date. Association of biliary tract disease with echinococcus cyst of the liver was noted by Klauber,[23] Sendler [9] and van Wijhe and Hammer.[24] In Klauber's case, the echinococcus cyst was discovered at a second operation after biliary disease had been proved at an operation two years previously. The same sequence held for Sendler's case, but in van Wijhe and Hammer's case the order was reversed, the echinococcus disease preceding the gallstones by several years and perhaps having no relation to the bronchobiliary fistula.

Oetiker [25] reported a most unusual bronchobiliary fistula due to cancer at the head of the pancreas and tuberculosis of the liver and biliary passages.

Bruhl and Lyon-Caen [26] recorded a case due to syphilis—the breaking down of gummatous tissue with the establishment of a fistula.

Lebert [18] found ascaris worms blocking the main bile ducts and causing abscesses of the liver and a fistula. Lobstein's [27] case, which

21. Eschenhagen: Deutsche med. Wchnschr. **28**:538, 1902.

22. MacDonald, G. C.: Lancet **2**:973, 1890.

23 Klauber, O.: Arch. f. klin. Chir. **82**:486, 1907; ibid. **91**:364, 1910.

24. Van Wijhe, S. J., and Hammer, E.: Nederl. Tidschr. v. Geneesk. **67**:771, 1923.

25. Oetiker, L.: Schweiz med. Wchnschr. **56**:501, 1926.

26. Bruhl, M. J., and Lyon-Caen, M. L.: Bull. et mém. Soc. méd. d. hôp. de Paris, 1909, p. 295.

27. Lobstein, quoted by Oliani: Complem du dict. d. sc. méd., vol. 34.

was similar, was accompanied by a purulent, not a biliary, sputum, and will not be included.

Trauma is the causative factor in some cases. Graham [6] reported one case which followed the kick of a horse; Tyrman,[28] and Elliott and Henry [29] cited cases due to gunshot wounds. In Stumpff's [30] patient, a previous biliary tract disease and diaphragmatic pleurisy were quiescent until the patient was knocked over by a bicycle, after which a bronchobiliary fistula formed.

A definite causative agent could not be assigned to the cases of Dmitrenko,[31] Laboulbène,[32] Smitten,[10] or Yates.[33]

In summary, cases of biliary bronchial fistula are most frequently due to echinococcus disease of the liver and to tropical (amebic) abscess of the liver. Next in frequency follows suppuration due to biliary tract disease—cholelithiasis or cholecystitis—the abscesses forming primarily in the subphrenic space, liver, gallbladder or biliary passages. Suppuration in these situations following any other etiologic factor which acts as a block to the normal outflow of bile may be effective in producing such a fistula: gumma, cancer, tuberculosis and ascaris worms. Trauma may act as a cause without the necessity of coincident infection taking place. In some instances, a definite etiologic agent cannot be assigned.

PATHOLOGY

In the establishment of a fistulous tract from the liver to the bronchi, certain conditions must be fulfilled. There must be a partial or complete interference with the normal bile passages, forcing all or a portion of the bile to seek exit by an unusual channel. Adhesion between the liver and the diaphragm, and the diaphragm and the base of the lung is necessary if the process is to be localized, and not a peritonitis or an empyema. A potential cavity must exist, either as an abscess, or as a walled-off space; or as widely dilated biliary or bronchial ducts. It is essential that fairly large bile ducts communicate with this cavity or directly through the fistula with the bronchi. Pressure of the contents of the abscess causes a weak spot in the diaphragm where the muscle fibers become separated and atrophic. The fistulous tract itself may be large or very small, direct or tortuous, but it must consist of firm fibrous tissue which tends to keep the passage patent and prevents it from being closed readily by débris. In almost every case in which a postmortem examination has been made, all these conditions

28. Tyrman, J.: Arch. f. klin. Chir. **89**:434, 1909.
29. Elliott, T. R., and Henry, H. G. M.: Brit. M. J. **1**:9, 1916.
30. Stumpff, J. E.: Nederl. Tijdschr. v. Geneesk. **1**:232, 1913.
31. Dmitrenko, L. F.: Vrach. Gaz. **16**:1 and 39, 1909.
32. Laboulbène, A.: L'union méd. **20**:271, 1875.
33. Yates, A. G.: Brit. M. J. **2**:1117, 1922.

have been present. The bronchial tree generally shows surprisingly little reaction to the bile which passes through it. The bile itself may be heavily infected or almost sterile, depending on the underlying pathologic process.

The most common localization for a bronchobiliary fistula is through the dome of the diaphragm over the uppermost part of the convexity of the right lobe of the liver. The portion of the lung involved depends entirely on the localization of the process below the diaphragm, and although it is usually posterior or lateral, it may be anterior in cases in which the gallbladder itself is adherent to the diaphragm (Mandard).[12] In Burgess'[34] case there was a remarkable variation as the fistula extended from the left lobe of the liver to the lower part of the left lung. Purulent collections may take extraordinary courses in trying to reach the exterior (Cayley,[2] abscess of the left lobe of the liver to the left pleural cavity; Legg,[3] through the diaphragm into the pericardium and the pleura, and Simmons,[35] in the liver, through the diaphragm into the middle mediastinum and then to the right bronchus).

The reaction in the tissues about the fistula is the same as that of any chronic suppurative process. There is always an infiltration with fibrous tissue and round cells, in addition to the products of acute inflammation when it still exists. The liver cells about the fistula show atrophy, and interstitial proliferation of connective tissue is prominent. The lung tissue near the fistula may show a catarrhal or purulent pneumonia (Schultze),[36] (Scevos Zervos),[37] edema (Dreschfeld)[38] or even a gangrene under some circumstances (Tuckwell),[39] but the acute process usually subsides with the drainage through the bronchus, leaving only a fibrous reaction about the fistula. When the process becomes chronic, there is little likelihood of an acute pulmonary flare-up.

Occasionally a cavity exists in the pulmonary tissue (Luzatto,[40] Mandard),[12] sometimes multiple cavities (Schultze)[36] or multiple abscesses (Schlesinger).[41]

SYMPTOMS AND SIGNS

Bronchobiliary fistulas give a clearcut picture which is unlike that of any disease process.

In the course of an illness pointing to the right upper quadrant of the abdomen or to the base of the right lung, a sudden critical change

34. Burgess, A. H.: Brit. J. S. **9**:253, 1921.
35. Simmons, D. B.: Am. J. M. Sc. **74**:463, 1877.
36. Schultze, F.: Virchows Arch. f. path. Anat. **61**:131, 1874.
37. Scevos Zervos: München. med. Wchnschr. **48**:147, 1901.
38. Dreschfeld, J.: Lancet **2**:867, 1879.
39. Tuckwell, H. M.: Tr. Path. Soc., London **21**:223, 1870.
40. Luzatto, M., quoted by Oliani: Cong. di med. intern., Roma **4**:432, 1891.
41. Schlesinger, H. L.: Mitt. a. d. Grenzgeb. d. Med. u. Chir. **16**:240, 1906.

occurs. The condition up to this time has been regarded as hepatic disease of some kind (cholecystitis, cholelithiasis, abscess of the liver, cholangeitis, etc.) or diaphragmatic pleurisy or pneumonia (stage of onset). Then a violent persistent pain usually occurs in the hepatic region, accompanied by chills and fever with or without jaundice (stage of blocking of ducts and pressure of accumulation in cavity). After a few days, the intense pain suddenly disappears with the onset of a racking, tormenting fit of coughing and the expectoration of a considerable quantity of bile stained material (stage of breaking through and formation of fistula). Some patients complain a great deal of a sense of oppression and suffocation, and a few of hiccups just before and during the breaking through.

There are a few exceptional cases in which the patient began to cough up bile without indications of previous disease (Burgess,[34] Laboulbène,[32] MacDonald [22]).

In the traumatic cases, there are usually signs of hemothorax or empyema preceding the expectoration of bile.

After the breaking through into the bronchus, the patient suffers with a cough which comes on in "paroxysms," "bouts" or "unbroken fits" and is described as 'incessant," "persistent," "troublesome," "tormenting," "irritating," "distressing," "hacking," "racking," "dry" and "hard." In only one case was it considered "very slight and soft" (Roper).[42] The cough is a serious symptom in most cases, giving the patient little rest, either day or night, and leaving him in a state of exhaustion. The patient quickly finds out that position influences the frequency of the coughing fits, and he assumes that position which reduces his cough to a minimum. A half sitting or erect position was assumed in a number of cases to prevent the irritating cough or the sensation of choking (Adam,[43] Dmitrenko,[31] Dreschfeld,[38] Faucon,[44] Graham,[6] Morton and Phillips, Perrier,[45] Roper,[42] Smitten,[10] Smith and Rigby [46]). In Thiroloix's [47] patient, the right lateral decubitus was impossible because of pouring out of bile: in Peacock's [15] second case also, the patient could not lie on the right side, whereas in his first case, the patient could not lie on the left side.

Apparently the size and direction of the fistula and the nature of the contents of the cavity influence the ease with which the expectoration is brought up. In Graham's [6] case, the patient had to "cough several times

42. Roper, A. C.: Practitioner **89**:718, 1912.
43. Adam, J.: Brit. M. J. **1**:836, 1890.
44. Faucon, V.: J. d. sc. méd. de Lille **2**:581, 1880.
45. Perrier, S.: Thèse de Lyon, 1900.
46. Smith, L., and Rigby, H. M.: Brit. M. J. **2**:313, 1903.
47. Thiroloix: Bull. Soc. clin. de Paris, 1888, p. 168.

before bile came up, and it never came up without coughing." Schlesinger's [41] patient brought up "only a little with great effort." On the other hand, there was a welling up almost without effort in the cases of Dreschfeld,[38] Klauber,[23] Laboulbène,[32] Roper [42] and Smith and Rigby.[46] Dmitrenko,[31] furthermore, claimed that "at night during sleep fluid comes out without the patient's knowledge." In MacDonald's [22] case, "the fluid came easily when he leaned forward"; and Oliani [8] said that "stooping down especially causes expectoration."

Once the fistula is established, the course is variable. Some fistulas remain open permanently, with a continuous discharge of bile through the bronchial tree. This usually indicates a complete block of one of the main ducts below. Mayo-Robson [48] recorded a case in which one of the hepatic ducts was blocked and in which there had been a steady expectoration of bile for nine years. A considerable number of instances of continuous expectoration over a period from two to six months are recorded. Other fistulas open and close in repeated attacks. A typical example is the patient of Ido and Yasuda [7] who had recurring attacks of one to two weeks' duration at least four times. Graham's [6] patient had an attack lasting sixteen days; he had a free interval of four months, and then a second attack of four to five days followed by a free interval of ten years before the fistula again opened for a long period. There are a good many reports of one attack succeeded by a free time and then a recurrence. Eichler [49] recorded a case in which the patient was free from trouble for twenty years between attacks.

The character of the expectoration often undergoes a cycle of change from a thick, purulent green, brown or yellow to a clearer liquid yellow or green, and finally to a thin, white mucoid type (Schultze,[36] Sendler,[9] Vissering [50]).

The amount of sputum expectorated daily varies from 50 cc. to over a liter. The average patient, however, does not maintain a constant level of expectoration, variations of from 100 to 200 cc. daily being frequent. In the average well established case, the patient coughs up at least 500 cc. daily. Tyrman's [28] and Nermord's [51] patients expectorated 1,200 cc. on one day and Oetiker's [25] patient, 1,100 cc. Roper's [42] patient averaged from 1 to 1½ pints (473.18 to 709.76 cc.) over nine years. The quantity raised with one coughing spell varies from 5 to 10 cc. Graham's [6] first patient coughed up "a large tablespoonful" at each bout of coughing. Perrier's [45] patient coughed up "small mouthfuls" of material.

48. Mayo-Robson, A. W.: Practitioner **75**:12, 1905.

49. Eichler, F.: Berl. klin. Wchnschr. (Ewald Festnumm) 1905, p. 97; Mitt. a. d. Grenzgeb. d. Med. u. Chir. **16**:551, 1906.

50. Vissering, E.: München med. Wchnschr. **43**:567, 1896.

51. Nermord, H.: Thèse de Paris, no. 239, 1891.

MacDonald [22] noted that the expectoration of bile increased from one half to one hour after the taking of food; and Graham [6] observed a similar phenomenon in his second case after doses of ¼ grain (0.015 Gm.) of mild mercuric chloride.

In gross appearance, the expectorated material shows considerable variation. The color may be bright or deep brown, grass green, "egg yolk" color or bright yellow or combinations of the foregoing colors. It may be thick and purulent, thin and serous or foamy mucus. There may be a homogeneous appearance or globes of mucus floating in the thin fluid. At times distinct layering is noted when the material stands in a container (Dreschfeld,[35] Schultze,[36] Tyrman [28]).

Occasionally large particles of broken-down tissue, old clots or purulent débris is present. Echinococcus cysts and hooklets have been found frequently. Gallstones rarely are expectorated (Colvée,[52] MacDonald,[22] Vissering [50]).

Microscopic examination may show masses of leukocytes, eosinophils, fat droplets, lung and liver cells, elastic fibers, squamous epithelium, fatty acid crystals, hematoidin crystals, bilirubin and "clumps of destroyed bile in yellow flecs" (Cattani,[53] Eichler,[49] Elliott and Henry,[39] Eschenhagen,[21] Fouché,[54] Ido and Yasuda,[7] Schultze [36]). Search should always be made for pieces of echinococcus cysts or hooklets, amebae, tubercle bacilli and neoplasm cells.

Chemical examination will give a certain diagnosis by any of the well established bile tests (Gmelin, nitric acid, mercuric chloride, Obermayer's reagent [Kapsinow's test]).

The patients often complain of the bitter taste of the sputum; sometimes there is a foul odor as well. The reaction of the material is alkaline, thus distinguishing it from vomitus.

Coincident with the appearance of the biliary expectoration, there may be a diminution and a disappearance of a jaundice. On the other hand, the blocking of the fistula may lead to jaundice and a painful tumor in the region of the liver. The establishment of a spontaneous fistula between the biliary passages and the intestinal tract may cause the spontaneous cure of a bronchobiliary fistula (Kehr).[55] The presence of a block in the common duct can be determined even in the presence of a bronchobiliary fistula by examination of the stools for bile.

Physical signs present in cases of bronchobiliary fistula vary from extensive frank consolidation at the base to a few scarcely audible râles

52. Colvée, quoted by Graham: Med. mod. Par. **1**:405, 1889.

53. Cattani, quoted by Graham: Gaz. med. ital. **45**:305, 315 and 326, 1888.

54. Fouché: Thèse de Paris, no. 338, 1885, p. 38.

55. Kehr, H., quoted by Oliani: Chirurgie der Gallenwege im Neue Deutsche Chirurgie, vol. 8, p. 240.

over a very small area—chronic bronchitis. Signs of a cavity are sometimes made out, but more frequently they are masked by the position. The dulness of the liver usually is increased, and the liver may be sensitive on pressure; occasionally a friction rub can be made out. In some cases the signs of subphrenic abscess are present.

DIAGNOSIS

The constant presence of bile in the sputum is sufficient to establish a diagnosis of bronchobiliary fistula. Graham [6] said that the expectoration might be bile-stained in jaundiced patients who developed either bronchitis or pneumonia, but that the amount and purity of the bile in a fistula makes the diagnosis certain.

It is important, however, to try to determine the etiologic factor responsible. Amebic abscess usually develops after exposure in the tropics. A period of dysentery with chills, fever and sweating and a painfully enlarged liver follow. The material expectorated is brick red and should be examined for amebas—on a warm stage; and by animal inoculation (kittens). Emetine acts as a specific. Instances of amebic abscess of the liver without preceding dysentery are not rare.

Echinococcus should be suspected in Greeks, Albanians, Italians and Spaniards, and in all people in a subtropical or tropical environment who have been in contact with sheep and sheep dogs. Additional aid is given when eosinophilia or an urticarial reaction follow rupture of a cyst. Positive complement fixation will clinch the diagnosis when an antigen is available. The sputum should be searched for hooklets or portions of walls of the cyst.

Most other cases give a typical picture of hepatic colic and may be classified as diseases in the liver or bile passages. It is important to know whether the common duct is obstructed completely or whether it is free. This is determined by examination of the stools for bile. The finding of gallstones in the stool is a decided aid in diagnosis (Felix,[56] Gaston [57]).

Roentgen-ray examination may give a great deal of information, especially if it is combined with the tetra-iodophenolphthalein test. The position of the fistula may sometimes be made out by anterioposterior and lateral fluoroscopy. Escudero, Terrada and Gallino [58] used iodized oil 40 per cent successfully by injecting 10 cc. into abscesses of the liver associated with bronchobiliary fistulas. They reproduced some excellent roentgenograms to show the outline of the abscess and the fistula, dif-

56. Felix: Cor.-Bl. f. Schweiz. Aertze **33**:250, 1903.

57. Gaston, J. McF.: Brit. M. J. **1**:325, 1885.

58. Escudero, P.; Terrada, H. M., and Gallino, M. M.: Rev. de la Soc. de Med. Int. **1**:563, 1925.

ferent postural positions being used to bring out the whole relationship. Oetiker [25] injected iodized oil from above through the bronchi, but was not able to demonstrate the fistula through the diaphragm.

PROGNOSIS AND TREATMENT

In some cases, the process is rapid and fulminating, the patient surviving the break through for only a short time. Death in these instances may be due to pneumonia, gangrene of the lung, general sepsis or hepatic insufficiency following prolonged jaundice (Adam,[48] Bristowe,[14] Carter,[59] Gaston,[57] Tuckwell,[39] Weiler [60]).

Other patients progress to a chronic state with continuous discharge of bile over long periods of time. In such cases there must also be some bile emptying into the gastro-intestinal tract, either by free ducts or by spontaneous fistulous openings. Still other patients have recurring attacks with reopening of a closed bronchobiliary fistula, sometimes after a very long closed interval (Graham [6] case one, Eichler [49]). It is unwise to count on the probability of a spontaneous cure when the condition has become chronic, because approximately 50 per cent of such patients ultimately succumb to some complication. There is no doubt, on the other hand, that there have been a fair number of cures from expectant treatment alone. The patient in the meantime goes through an agonizing period until the cure is effected by ulceration between the biliary passages and the gastro-intestinal tract or by the expulsion of the obstructing material through the sputum (Cattani,[53] Colvée,[52] Faucon,[44] Felix,[56] deHavilland Hall,[61] Kehr,[55] Vissering [50]).

Operative treatment under modern conditions promises relief in a large proportion of cases. There are enough excellent results to warrant advocating operation in all such patients whose condition allows it (Burgess,[34] Elliott and Henry,[29] Graham [6] case two, Klauber,[23] Körte,[62] Mayo-Robson,[45] Morton and Phillips, Roper,[42] Sendler,[9] Smith and Rigby,[46] Tyrman,[28] Yates.[33] Equally good results have been obtained in cases of echinococcus. Even patients in a serious condition can be carried through operation in carefully worked out stages. Many operative failures in the past were due to poorly conceived technical procedures and too extensive operation which led to death from shock. It is of the utmost importance to attempt to estimate the probable pathologic process before undertaking any therapy. It is equally important to gauge the

59. Carter, G.: Brit. M. J. **1**:1119, 1889.

60. Weiler, K.: Wien. klin. Wchnschr. **15**:353, 1902.

61. Hall, F. deHavilland: Brit. M. J. **2**:924, 1886; Proc. Med. Soc., London **10**:69, 1887.

62. Körte, quoted by Eichler: Beitr. z. Chir. d. Gallenwege u. Leber, 1905, vol. 7.

patient's resistance and to plan accordingly. Every case of broncho-biliary fistula offers a different problem, and the decision as to whether the operation should be performed by the abdominal or by the transpleural route depends entirely on the results of the examination. We cannot agree with Smitten [10] that the transpleural approach is the method of choice. The abdominal approach can be carried out under scopolaminemorphine and procaine hydrochloride anesthesia, and one procedure may be all that is necessary.

If the probable block can be ascertained beforehand and the condition warrants it, operation to remove the block (stone in common or hepatic duct, echinococcus cyst or other obstruction) may be all that is needed to cause spontaneous healing of the fistula.

If the condition apparently is dependent on a subphrenic abscess, we advocate the approach by transposing the pleural sac upward. Because the majority of these patients are greatly distressed when they lie down and because they cannot take general anesthesia, we advocate the exposure used in our case. It can be carried out as simply as a transpleural operation with the patient sitting upright and by the use of procaine hydrochloride anesthesia. The advantage is that the uninfected pleural sac can be easily identified and mobilized out of the way, thus safeguarding it from infection. In addition, the patient who is already worn out, is not subjected to the shock of a pneumothorax. The fistula or abscess is approached through the diaphragm, the adhesions to the liver and the diaphragm being gently separated. A whiff of nitrous oxide or ether may be necessary during this manipulation. The adhesions between the diaphragm and liver are a sufficient safeguard against soiling the peritoneal cavity. Drainage is dependent. The danger of secondary pneumothorax must be kept in mind, and suction to the pleural cavity used in case this occurs.

CASES REPORTED IN THE LITERATURE

ADAM'S CASE.[42]—A woman, aged 70, had had obstructive jaundice with pain seven years before the case was reported, and a similar attack without jaundice two years before. In January, 1889, more or less persistent obstructive jaundice developed. The pain was never great. Suddenly, on July 13, 1889, a severe persistent pain in the hepatic region and rigor developed and continued the next day. The pain suddenly ceased on the morning of July 15 with the onset of cough and profuse expectoration. Physical examination revealed an exhausted, thin, woman with jaundice, sitting upright and refusing to lie down because if she did, she had a distressing intermittent cough accompanied by expectoration. There was acute bronchitis in the right lung, less marked in the left. The sputum was thick, greenish and yellow with a slight admixture of air. Careful examination showed it to be bile. The temperature was normal. The abdominal examination was unsatisfactory. During the next four days the urine and the conjunctivae became lighter colored, but the stools remained clay-colored. The pain in liver had disappeared. Expectoration of the same fluid continued until the evening of

July 18. On the morning of July 19, the right lung had become solid. The temperature remained normal. The patient gradually became weaker, more distressed, and more deeply jaundiced and swelling developed in the region of the gallbladder. On July 26, this could be palpated as a sensitive cyst containing fluid. The patient became progressively worse, and died on July 30. Autopsy was not performed.

BRISTOWE'S CASE.[14]—A woman, aged 32, was admitted to the hospital on April 20, 1852, and died on June 9, 1852. She suffered from intense jaundice and other symptoms of hepatic disease, and shortly before death spat up bile-stained pus. Postmortem examination revealed a deeply jaundiced patient. The left pleura was universally adherent; in the right pleura there were a few adhesions at the posterior part and the base of the lung was firmly attached to the diaphragm. The upper and middle lobes were healthy and crepitant; the lower were airless from collapse, tough, fibrous and slate colored. In the center of the base of the

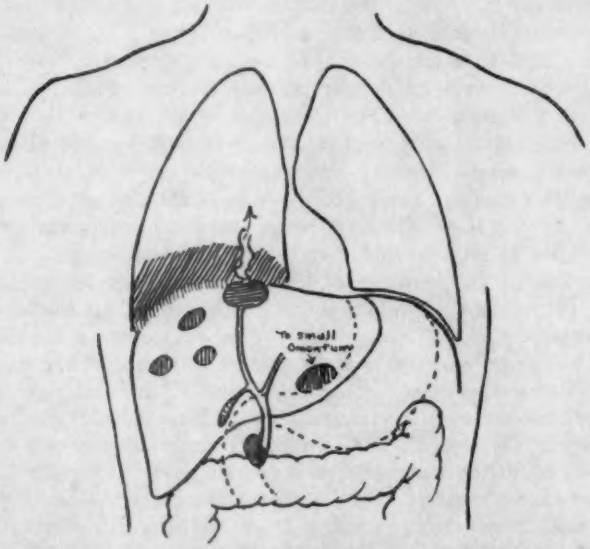

Fig. 2 (Bristowe's case).—Collapsed lower lobe of the right lung; liver full of abscesses, one of which opened in the bronchus; common duct blocked by a stone.

right lung was a superficial, eroded and flocculent area of about 2 square inches; this corresponded to a perforation in the diaphragm and a cavity in the liver. The contents of the bronchial tubes connected with the lower lobe consisted partly of biliary matter, and one tube was directly traced to its opening on the ulcerated surface. The liver was much enlarged and filled with cavities of varying size, the largest being the size of hen's egg. The largest cavity on the convexity of the right lobe of the liver communicated with the lung. There was a large ragged opening in the diaphragm. The cavity on the under surface of the liver in the small omentum communicated with both the duodenum and the colon by a ragged, ulcerated opening the size of the little finger. The common duct was blocked by a hard, globular, rough stone the size of a large marble, but there was a trace of bile below this in the duct. The gallbladder was small. The common duct ended in a cavity in the omentum. There was also a biliary fistulous communication with the branches of the portal vein.

BRUHL and LYON-CAEN'S CASE.[20]—A woman, aged 33, was admitted to the hospital on Nov. 20, 1908. She had complained of an increase in the size of her abdomen for six months and expectoration of increasing amounts of yellow-green material of markedly bitter taste. She had had typhoid at the age of 5, scarlatina and syphilis at 19. Eight years before admission she had icterus of two months' duration. In April, 1907, painful crises simulating hepatic colic came on, but although she was subicteric, there was bile in the stools; from this time, she knew she had an enlarged liver. She also had had respiratory trouble and cough for five years. Two years before the case was reported she had marked hemoptysis, and slight hemoptysis had occurred from time to time since then. She lost weight and expectorated habitually. In October, 1908, she expectorated a large quantity of greenish viscous liquid, leaving a prolonged bitter taste and the sensation of having emptied a thoracic collection. A basinful of biliary sputum was expectorated daily in small amounts. Then the quantity of bile progressively diminished; at the end of four days, the sputum was scarcely tinted green. Physical examination revealed a pale, thin patient without fever. The sputum was mucopurulent, with a clear green color. There were no elastic fibers or tubercle bacilli, but large numbers of polymorphonuclears were found. The biliverdin test was positive (Grimbert). The right side of the thorax showed a zone of dulness, continuous dulness of the liver; rough respiration and râles were heard and diminished respiration below. In summary, there were signs of bronchitis without signs of a cavity. The veins about the umbilicus were distended; there was ascites and the liver was very large, hard and irregular, and painless, extending for five to six fingerbreadths below the costal margin. A hard tumor the size of an orange was found in the left lobe; it was not fluctuant, but painful. Icterus was not present. The spleen was enlarged. The stools were almost normal. Eosinophilia did not occur. The echinococcus fixation reaction (Weinberg) was negative. The quantity, nature and color of the sputum varied; sometimes it was mucopurulent, sometimes green. The chest signs also varied; after a large amount of expectoration, the dulness in the right side of the chest diminished. On Dec. 23, 1908, a roentgenogram showed two opaque zones, one was posterior to the right side of the chest near the vertebral column, the other anterior to the stomach. On Jan. 8, 1909, about 500 Gm. of yellow ocher liquid with reddish reflection, recalling fresh bile was expectorated during the night. It had a very bitter taste. On January 30, Gosset performed an exploratory laparotomy. An enormous Spigelian lobe was found, but no pus nor cysts. Death occurred eight days later from hepatic insufficiency. Postmortem examination revealed a large liver, irregular and multilobular. Whitish masses projected from the surface in three different regions; they were hard, not depressible not fluctuant, and fluid was not found on aspiration. One mass the size of the fist in the region of the right lobe formed the floor of a subphrenic cavity about 6 cm. in diameter, the superior wall corresponding to the midportion of the diaphragm, which was perforated at certain points, adherent to lung tissue and sclerosed at others. The walls of this cavity were stained green by bile. There were two other gummas; one was the size of the palm of the hand, and was located on the edge of the left lobe, the other was the size of a nut, and was found on the posterior face of the liver. The right lung was much reduced in size, and its surface was irregular, hard, sclerosed, and intimately adherent to the diaphragm. It was difficult to find a fistula communicating with the bronchus. The condition was probably syphilis of the lung developing simultaneously with gumma of the liver.

Summary.—The probable course of the condition was as follows: (1) icterus eight years before admission, due to gumma of the liver; (2) cough and hemoptysis three years later, and invasion of the lung by gumma; (3) a painful syndrome like hepatic colic and opening of the hepatic canal by gumma and (4) expectoration of bile and formation of a fistula.

BURGESS'S CASE.[84]—A woman, aged 59, was admitted to the hospital on May 5, 1921. On Dec. 2, 1920, while walking home from her office she had a sudden attack of coughing and expectorated some thick white phlegm. This recurred frequently during the next three days, and on December 5, she noticed that the expectoration was yellow and had a bitter taste. Except for a few short periods of two or three days each, during which the sputum was white, frothy and devoid of any bitterness, she had expectorated daily a frothy yellow fluid varying in quantity from 10 to 20 ounces (295.73 to 590.47 cc.). The sputum showed pure bile without pus or blood cells or tubercle bacilli. *B. coli* was present on culture.

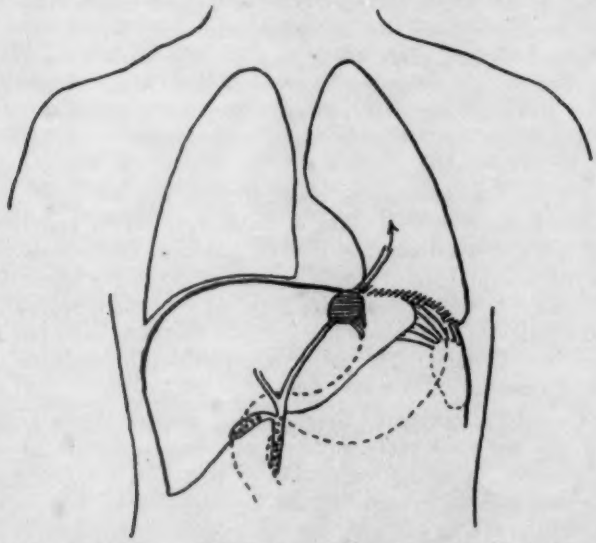

Fig. 3 (Burgess' case).—Gallbladder and common duct full of stones; an abscess cavity in the left lobe of the liver connecting with the left hepatic duct with a break through into the left bronchi.

The only previous illness that she could recall occurred about four and a half years previously, when she had had a headache and had vomited bile; this illness had kept her from work for only a day and a half. Abdominal pain or jaundice did not occur. At the beginning of her illness, she lost considerable weight, but for the last three months her weight had remained constant. The results of the examination were normal. The expectoration varied in color from yellowish green to bright yellow, was alkaline in reaction and frothy; the sputum was coughed up in quantities of 1 drachm (3.69 cc.) every ten or twenty minutes. Frequent coughing allowed her little rest at night. On May 28, 1921, exploratory laparotomy was performed; the gallbladder was found to be contracted and thick-walled, and calculi were found in the gallbladder and in the dilated common duct. The right lobe of the liver was not adherent to the diaphragm, but the left lobe was everywhere adherent, as was also the cardiac part of the stomach and the spleen. The margin of the left lobe was inseparably incorporated with a

rounded, stony hard swelling the size of a golf ball and was densely adherent to the diaphragm above, to the spleen posteriorly and to the left, and to the lesser curvature of the stomach almost to the cardiac orifice. On chiselling through the wall of this swelling, some bile and a quantity of "biliary mud" escaped. The adhesions were freed and the mass removed. An actual opening was not seen through the diaphragm. The common duct was opened and twelve calculi were removed. A probe passed in the left hepatic duct could be felt under the excised area of the left side of the liver. The gallbladder with several small calculi was removed. Recovery was uneventful. The frothy white expectoration steadily diminished and was free from bile for the first ten days. The patient left the hospital one month after operation, free from cough and expectoration.

CARTER'S CASE.[50]—A woman, aged 70, was in good health except for an occasional "spasm" until two years previously, when she had well marked biliary colic followed by jaundice. Attacks continued at intervals of five or six months. In July, 1888, a severe attack occurred with permanent jaundice and clay-colored stools. One month later, she had an attack resembling bronchitis, in which she daily expectorated about 2 pints (946.3 cc.) of thin, dark green biliary matter. Large moist râles were heard at the base of the right lung. Nothing was felt in the abdomen. Although the expectoration occasionally ceased, it recurred, and the patient gradually wasted and died. Postmortem examination was not performed.

CATTANI'S CASE[51] (according to Graham).—A woman, aged 48, who was healthy previously, suffered with pains in the hepatic region and from digestive disturbances, and afterward coughed up bile and pus. Sometimes the tissue of the lung and the liver and striated muscle were found in the expectoration. She suffered from jaundice, and a tumor was noted in the hepatic region. An inflammatory process followed by suppuration began in the liver and extended through the diaphragm into the lung. The patient recovered. The probable cause of the condition was a stone in the common duct.

COLVÉE'S CASE[52] (according to Graham).—A general in the army who had been in the tropics for some years, suffered with hepatic disease, presumed to be the result of malaria. After having attacks of bronchitis for four years, he was seized with severe pain in hypochondrium, accompanied by fever and vomiting. He commenced to cough up bile after some days and continued to do so for eight months. On several occasions, he coughed up small biliary calculi, which fell into the spittoon with a distinct noise.

DMITRENKO'S CASE.[53]—On Oct. 8, 1907, a man, aged 50, complained of cough and expectoration of yellow liquid. He became sick three years previously with cough and pains in the left side. At first there was blood in the expectoration, and then it became yellow. One year before the case was reported, there was an increased quantity of yellow sputum. Two months before examination, edema of both legs occurred. Five years before, he had had an ulcer of three weeks' duration on the left leg. Examination revealed poor nutrition; loss of weight and edema of the legs. On respiration, the left side of the chest lagged. The percussion note was higher in pitch on the left above and below the nipple. The breathing below the left scapula was amphoric. Dry râles were heard in the same area and also in the region of the right nipple. A splashing sound was heard in the region of the umbilicus. The patient could not distinguish a bitter taste. His appetite was satisfactory for all kinds of food. There were bulky, uncolored stools daily. The liver was normal in size and position but was not tender. A specimen of urine, 400 cc., was normal. The sputum was tenacious and stringy; it was comprised of 98.2 per cent of water and 1.8 per cent of solids; these solids

consisted of 1.25 per cent of organic matter and 0.55 per cent of inorganic matter. Bile acid and pigments were present, and only occasional epithelial cells; it was negative for tuberculosis bacilli. On October 9, the stools were still colorless. The severe cough was relieved by codeine. The cough occurred when the patient was lying on the right side, and much yellow fluid was expectorated. At night the fluid escaped while the patient was asleep. The edema of the legs subsided. On October 13, the stools were bright green. The following amounts of sputum was expectorated: from 10 a. m. to 2 p. m., 130 cc.; from 2 p. m. to 4 p. m., 70 cc.; from 4 p. m. to 6 p. m., 50 cc.; from 6 p. m. to 8 p. m., 50 cc.; from 8 p. m. to 10 p. m., 30 cc.; from 10 p. m. to 12 p. m., 35 cc.; from 12 p. m. to 8 a. m., 175 cc., and from 8 a. m. to 10 a. m., 200 cc., making a total of 740 cc. On October 14, edema extended to the knees. There was a gallop rhythm of the heart. On October 15, 840 cc. of sputum was expectorated. Indican was found in the urine; the stool was colorless. On October 20, the urine was negative for urobilin and bile pigments. On October 21, the edema of the legs had disappeared; the heart was normal. The temperature varied from 36.8 to 39.9 C. (98.4 to 103.8 F.). On October 23, the stools were the same. On October 24, the cough was much lessened and on October 25, it had almost disappeared. On October 27, 400 cc. of sputum was expectorated, and on October 28, 600 cc.; the splashing sound disappeared. Anorexia developed. On October 29, there was 600 cc. of sputum; on October 30, 650 cc.; on October 31, 750 cc. and on November 1, 800 cc. The stools remained the same. On November 3, 800 cc. of sputum was expectorated. The patient was discharged from the hospital. Differential diagnosis was not made. Persistent bronchobiliary fistula was present. There was no indication of the etiology in the anamnesis or on examination. Nothing in the past history indicated gallbladder disease or echinococcus. No clue was found to the origin of the abscess of the liver. The stool was colorless, and there was no reaction to bile, although the patient was never jaundiced. The common duct must have been blocked. Although the patient lost weight, the stools became normal. Mild mercuric chloride was given as a cholagog and the amount of sputum became less. Râles were heard in the region of the fistula.

Dreschfeld's Case.[88]—A man, aged 62, was admitted to the hospital on April 23, 1879. He had been in good health until eight years before admission, when he had hemoptysis and was treated, with relief. Shortly after he had had an attack of acute inflammation of the bowels with great abdominal pain lasting for weeks. He was in perfect health in September, 1878, when he had another attack of pulmonary hemorrhage that soon subsided. Since then he had had more or less cough and expectoration. For some months, he had noticed that the expectoration was bright yellow. His general health began to fail and he was obliged to give up work. He was never jaundiced. Examination revealed a slightly emaciated man without jaundice, who was slightly dyspneic and had an irritating cough. Slight emphysema and bronchitis of both lungs were present. A large quantity of thick, viscid, frothy, deep green sputum was expectorated. On standing, the sputum separated into two layers; the upper layer was green and fluid, and the lower one, yellowish and thick. The lower layer consisted of pus cells, epithelial and granular cells, all tinged with bile-colored material, while the supernatant fluid gave all the reactions for bile. From 8 to 10 ounces (236.5 to 295.7 cc.) of sputum were coughed up daily without any great effort. When the patient was recumbent, the sputum rose in his throat and was brought up without coughing; hence he was obliged to sit up. The dulness of the liver was normal. The feces were normal also. The urine did not contain bile. The symptoms remained the same for some days; then on May 3, 1879, an attack of vomiting of bilious matter

occurred followed by profuse hematemesis. The physical signs remained the same. The urine now contained bile coloring matter, but no albumin or blood. Jaundice was not present. On May 7, the patient continued to vomit food, bile and blood. Bronchial expiration, subcrepitant râles and increased vocal resonance were heard in the right suprascapular region. In the right axillary region there were tympanitic percussion, weak vesicular breathing and mucous râles. On May 9, the patient was weaker. The physical signs remained the same; incessant hiccups developed and with every hiccup bile was raised. Twelve ounces (354.8 cc.) of sputum was raised in twenty-four hours. On May 13, the patient was much worse; the hiccups and sputum continued and he gradually grew weaker. He died on May 15.

Autopsy revealed the upper surface of the liver and the lower surface of the right lung bound down firmly to the diaphragm by old adhesions. The liver was normal in size and in appearance. The lower surface was traversed by numerous fibrous bands, one of which was stretched tightly across the neck of the gall-

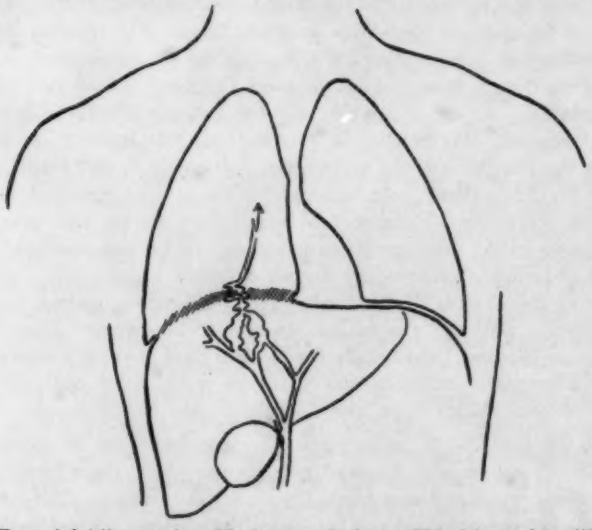

Fig. 4 (Dreschfeld's case).—Hydrops of the gallbladder with dilated hepatic ducts; the ducts filled with small stones and infected material; a break through the diaphragm into the bronchus.

bladder, obliterating the cystic duct. The gallbladder was dilated and filled with clear mucus without a trace of bile or gallstones. Cross-sections of the bile ducts showed some to be perfectly normal, while others were dilated and filled with thick, creamy, yellow fluid. These were traced to the upper surface of the diaphragm, and were found to be filled with the same material and with small branched calculi. From this cavity, a narrow tortuous canal led into the lung and bronchus. The walls of the fistula were of firm fibrous tissue. The lung about the fistulous tract was edematous and bile stained.

Eichler's Case.[40]—A patient, aged 43, twenty years previously had been suddenly awakened at night with an irritating cough and expectoration of a thin, fluid, yellow-green sputum. The cough with a greenish expectoration lasted unbroken for a half a year and then ceased. Since the beginning of June, 1905, there had been again a tormenting, irritating cough, with greenish sputum that tasted of bile. A diagnosis of bronchobiliary fistula was made. The patient

coughed up daily an average of from 700 to 900 cc. of almost pure bile. Masses of leukocytes, pieces of lung tissue and elastic fibers were found in the sediment. The urine was entirely free from bile; the stool was light colored, but contained hydrobilirubin (sublimate test). An operation was performed to close the fistula, but the patient died the day following operation from weakness of the heart and edema of the lungs.

At postmortem examination there was an open fistulous communication between the bronchiectatic cavity of the right lower lobe and an enlarged bile passage. The latter was one of the main branches of the right hepatic duct in the ramifications of which two gallstones the size of cherry stones were found.

EICHHORST'S CASE.[63]—An Italian man, aged 56, complained of cough and daily expectoration of from 200 to 500 cc. of greenish-brown bile. Examination revealed severe jaundice, dulness with diminished vocal fremitus in the right intrascapular space and tenderness in the region of the gallbladder. The trouble began in

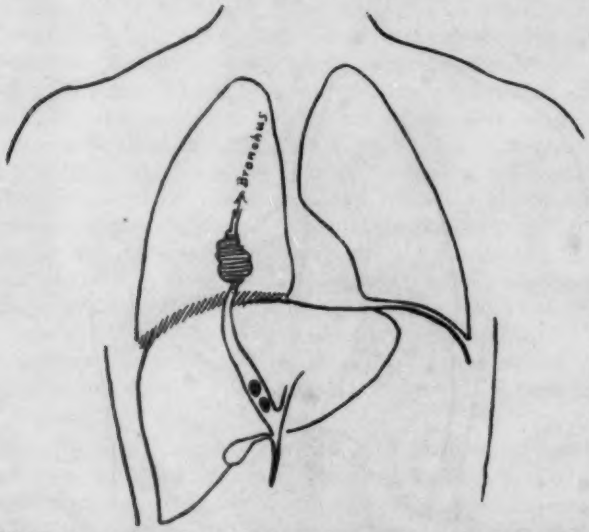

Fig. 5 (Eichler's case).—Bronchietic cavity in the lower lobe of the right lung with a fistula connecting from the right hepatic duct which was blocked with stones.

October, 1901, with jaundice without apparent cause. In August, 1902, he began to cough, and four weeks later he expectorated biliary fluid. There had never been pain. The stool contained bile. An exploratory puncture of the right pleural cavity did not reveal any fluid even though the puncture was 10 cm. deep. The condition was thought to be due to gallstones. Operation was refused.

ELLIOTT and HENRY'S CASE.[20]—A septic hemothorax of the left side was caused by a shrapnel ball, which traversed both sides of the chest. At some time after the wound was received, the patient began to expectorate sputum mixed with yellow bile, but the exact date of this could not be ascertained, because the symptoms had caused a psychical obsession which made all the patient's statements on the question unreliable. On the twenty-sixth day, the empyema on the left side was drained. It contained streptococci and gas bacilli. Frothy sputum with patches of

63. Eichhorst: Cor. Bl. f. Schweiz. Aertz **33**:792, 1903.

bright yellow bile and once or twice with fleshy masses of necrosed liver (proved by microscopic section) continued to be coughed up. Lieut. Col. H. P. Hawkins reported from England that the expectoration of bile ceased in the tenth week. The empyema sinus was practically closed at the fourteenth week, and the patient was rapidly becoming well.

ESCHENHAGEN'S CASE.[21]—A woman, aged 35, was admitted to the hospital on Sept. 10, 1901. For three years, she had had attacks of cramps on the stomach but they were always of short duration. In July, 1901, a severe attack occurred accompanied by jaundice. Four days before admission, she had a sharp attack with a chill. Examination revealed marked icterus; high fever; diffuse râles over the lung; abdominal distention; the liver sensitive to pressure, with a smooth surface and a firm, lower edge one fingersbreadth above the navel. No tumor was palpable in the region of the gallbladder. The urine contained bilirubin and a trace of albumin. The diagnosis was cholelithiasis complicated by

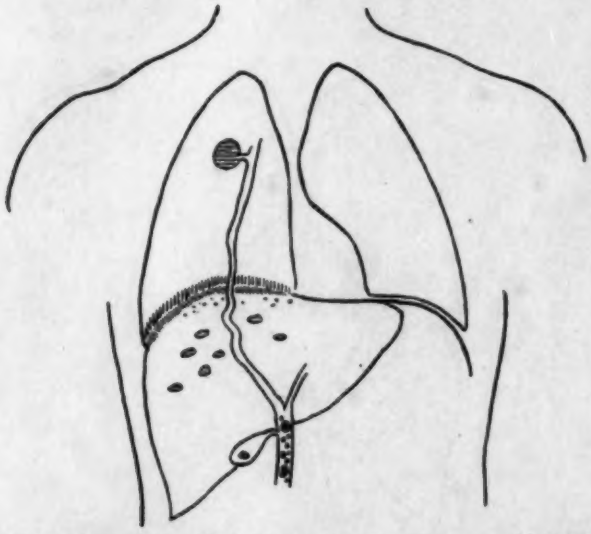

Fig. 6 (Eschenhagen's case).—Stones in the common duct with infection, abscess of the liver, fistula through the diaphragm, tuberculous cavity at the right of the apex and retrograde extension of the tubercules in the abdomen.

purulent cholangeitis. Operation was considered out of the question because of general septic infection. The liver was not palpable three days later; the dulness diminished, icterus was still intense, and the feces were entirely pale. The patient became comatose. No trace of leukocytes was found in the blood. During the coma there was dulness in the lower part of the right side of the chest posteriorly, and the breathing was bronchial. One week later, the icterus and coma gradually subsided. The dulness of the liver became normal. The liver was no longer tender, but the fever did not subside. Over the lower part of the right lung posteriorly, in a patch about a handbreath in width was intense dulness, bronchial breathing and loud, ringing râles. The sputum was abundant, slimy and purulent. On Sept. 20, 1901, it suddenly took on an offensive foul odor, which lasted five days; then the odor disappeared, but fever and abundant expectoration continued. The infiltration of the lower part of the right side of the chest disappeared gradually, leaving only a diffuse catarrhal condition. Since October 31, the

sputum had showed the color of an egg yolk; microscopically, there were some amorphous masses of bilirubin, hematoidin crystals, fatty acid crystals and amorphous material. At the end of November, the expectoration slackened, the fever disappeared, and only a few catarrhal râles remained. The patient felt better. A pleural tap gave only a serous exudate, bacteria-free. In the middle of December, the patient was suffering again with ascites, edema and fever. On the night of December 27, the patient again had a sudden abundant expectoration of golden yellow, clear, slimy, but not purulent sputum. Gmelin's test for bile was positive. The yellow color diminished again, but occasionally appeared. A slight general icterus remained. Ascites and weakness increased. The patient died on Jan. 17, 1902. Postmortem examination revealed 4 liters of cloudy, icteric fluid in the abdomen. The omentum was adherent to the liver. The peritoneum was red with gray submiliary tubercles. The pericardium was completely obliterated. The anterior, medial and lower surface of the right lung was adherent to the diaphragm. A fistulous opening led from the diaphragm to a dilated bronchus. An old, caseous, thick-walled cavity the size of a hazelnut was found in the upper lobe of the right lung. The bronchi showed purulent inflammation. The bronchopneumonic patches in the left lung were not tuberculous. Numerous thick adhesions were found between the liver and the surrounding organs. The diaphragm could be separated from the surface of the liver fairly easily. Its peritoneal surface showed a thick eruption of tubercles. On pressure on the common duct, a small stone sprang out into the duodenum, followed by dammed back bile. The common duct was dilated, and contained cloudy bile and two large and numerous small gallstones. The gallbladder was markedly atrophic and thickened; bile was not present, but there was a stone the size of a hazelnut. In the substance of the liver many small and one large fresh abscess were found as well as multiple small old abscesses of the liver. The spleen had great caseous tubercles throughout. The kidney showed hemorrhagic nephritis. The picture was one of an infected cholangeitis giving rise to multiple abscesses of the liver, one of which had broken through into the main bronchus of the right lung, causing gangrene and pleurisy.

Faucon's Case.[44]—A boy, aged 7½ years, was admitted to the hospital on May 29, 1878, with a complaint of pain in the stomach, fever, loss of appetite and loss of weight of some weeks' duration. On May 30, the abdomen was ballooned out; the liver was large and painful, and he had fever. On June 6, the liver had increased in size and was more painful. On June 21, the tumor was fluctuant. The patient's condition was serious. A puncture in the epigastrium evacuated 130 Gm. of pus. During the following days, the symptoms diminished. On June 27, the pocket refilled, but puncture did not give results. On July 12, the tumor diminished notably. On July 20, there was a loss of appetite; fever reappeared, and the pocket filled again. Cautery was used. On July 23, the child coughed considerably, but nothing was found on auscultation. On July 25, the condition became grave; the cough was marked. The parents took the child home against advice. Some days later, according to the mother, he vomited five or six times, from about twenty to twenty-five minutes after eating; there was also diarrhea. The cough was persistent, always frequent and painful, but sputum was not raised. The child could not rest on the back or the side. He raised himself continually and shifted his position, crawling from one bed to another in the hope of finding relief. Relief was obtained only on sitting on an armchair, supported by pillows. The cough was much less frequent and much less pronounced in this position. At the end of three weeks, he began to expectorate, little by little, and always after violent coughing. The sputum was of a different

character from the first, sometimes dark brick red, sometimes yellow and thick, and sometimes green. It had an insupportable odor, even annoying the patient himself. This lasted for about three weeks; on September 10, the sputum became dark green, and was much less odorous and less abundant; the cough was also less frequent. Diarrhea persisted, but from this time, stools suddenly became white. There was no icterus. The child complained of a bitter taste. Toward the end of September, the cough was much diminished, and vomiting and diarrhea had ceased; the stools were still decolorized, but less so than formerly. The patient was thinner, in spite of the return of appetite. He was pale and cachectic, with only the skin left on the bones. There was another attack of diarrhea. The stools were greasy and decolorized. After violent bouts of coughing, he expectorated yellow liquid mixed with aerated mucus. At times this liquid was ejected by vomiting. Signs of general bronchitis were present, consisting of subcrepitant fine and large râles, more over the base of the right lung, especially over the lower two thirds of the chest. There were no signs of tuberculosis. The liver was enlarged and painful. This condition continued for ten days, and then the expectoration suddenly diminished. The bouts of coughing also diminished progressively, and the child recovered his strength with rapidity and took on weight. On Dec. 21, 1879, he left the hospital completely healed, without the recurrence of expectoration of bile. On June 3, 1880, the expectoration had not recurred, and the condition was excellent. There was an epigastric hernia about the scar but no other pathologic condition. This patient showed all the symptoms of a suppurative hepatitis.

Felix's Case.[66]—A woman, aged 48, for three years had had short but severe colicky attacks without jaundice. In the middle of January, she had a sudden sickness with high fever and severe pain in the cecal region. This pain diminished when an ice bag was applied over night. Examination showed myoma and a smooth, convex slightly sensitive swelling under the right costal margin (this was mistaken for a kidney). After a few days, she was relatively well but on January 23, she suddenly developed fever of 39.8 C. (103.6 F.); her pulse became feeble and the rate rapid; she was prostrated, had a severe pain in the abdomen, and marked, foul bleeding from the uterus. The myoma and the surrounding abdominal wall appeared sensitive on pressure, but the region of the liver was not sensitive. Jaundice did not occur. The feces and urine were normal. The condition was thought to be an infection of the myoma with degeneration. On the following night, she had a fit of coughing which lasted five minutes; she raised some sputum, which was the color of the yolk of an egg and was uniform throughout. She complained especially of lack of rest on account of the irritative cough, but still more of the bitter taste of the sputum. Microscopically, the sputum showed pure bile and numerous pus cells. The bleeding from the uterus stopped, the swelling under the right costal margin diminished, the fever decreased and only rarely in the evening did the temperature become as high as 38 C. (100.4 F.). From January 24 to 28, in spite of treatment with codeine, morphine and other drugs, the cough with biliary sputum persisted with pauses of only from five to fifteen minutes, and prevented sleep. The stools had pigment in them until January 26, but from that time they became completely bleached, not gray, but a zinc white. The bile-free urine was a brick dust red. On January 28 and 29, she had an undisturbed sleep for six hours following the administration of morphine. There was loss of weight in spite of the fact that she ate an ordinary diet. The expectoration remained approximately the same until February 8, amounting to from 150 to 300 Gm. daily; at first it consisted of pure bile, but toward the last it was thinned out with bronchial and salivary

secretions. The stool was still completely colorless and smooth white; it first showed a slightly yellowish stain on February 9. It was carefully searched repeatedly for stones and pus, without results. On February 10, it became completely stained, and half formed; at the same time, a large three-facetted pigmented stone was found, the greatest diameter being 2.5 cm.; the smallest, 11 mm. The character of the sputum changed at once, no longer containing bile pigment; the quantity was not diminished, but became pure pus of a foul fecal type for the first time. There were marked râles over the lower lobe of the right lung anteriorly; the pleural sac remained free; and the temperature was normal. No more stones were found in the stools in spite of careful control up to February 19, but from then on several times great quantities of slimy pus was found. From this time, the cough and expectoration completely vanished; the patient quickly regained health and left her bed. At the present time, she is well. The size of the gallstones and the amount of foul pus apparently showed that healing had occurred by means of a gallbladder-duodenal fistula.

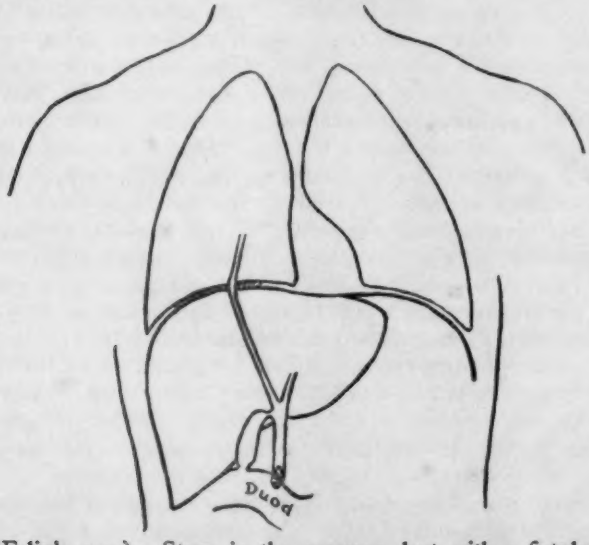

Fig. 7 (Felix's case).—Stone in the common duct with a fistula through the diaphragm; cure obtained by spontaneous cholecystenterostomy.

Foard's Case.[19]—General Twigg was taken sick, April 8, 1859, with a disease supposed to be bilious colic, for which he was treated. There was slight improvement until May 2, when he rode out in a carriage. Since April 16, there had been profuse perspiration during and after sleep and much debility and pain in the right shoulder. After a light dinner on May 7, he had pain in his stomach and vomited. On May 8, fever and chill developed and continued the following day. On May 9, the skin and conjunctivae were deeply tinged with bile. The patient loathed food. There was a thick coating on the tongue, bile in the urine, a slight cough and severe pain in the right shoulder that did not increase with pressure or motion, great debility and an "indescribable sense of suffering and oppression; he felt as though he were paralyzed"; the pulse rate was 100; the lungs were healthy, and the liver was normal on examination. Pain in the region of the liver did not occur at any time, but there were frequent chills and perspiration. On May 10, he slept till 4 a. m.; then a dry hacking cough developed and continued

until 7, when he suddenly vomited about a half pint of pure bile. He coughed constantly all day and passed bile; mucous râles were audible when the ear was applied to the chest; the respiratory murmurs were natural. The pain in the shoulder was not so great as on the previous day; frequent chills occurred during the day; the skin was hot or bathed with perspiration. On May 11, the feces were bilious. The cough and expectoration of bile continued all night. There was a slight diminution of bile in urine. Change did not occur in the skin. The patient had a severe pain in the shoulder during the morning, followed a few hours later by a great flow of bile and mucus; there were signs of a right hemothorax. The patient suffered greatly, not from pain, but from a sense of suffocation and impending dissolution. He was exhausted and his spirits were depressed. On May 12, there were cough and expectoration; the urine was of a more natural color; the skin was not so yellow; there was dullness over the lower part of the right lung posteriorly; the respiratory murmur was indistinct. On May 13, there were less bile and mucus, no appetite, and great debility. On May 14, the cough increased considerably; a pain developed in the shoulder the previous night. On the right side posteriorly, there were bronchial breathing and increased vocal resonance. The urine was natural and the icterus had nearly disappeared. On May 15, the cough and expectoration were diminished, the sweats continued; and the patient hiccupped occasionally. On May 16, the cough and expectoration continued; the mucus was mixed with bile; there was no pus; he had a hiccup and slight pain in the right shoulder. Early in the morning of May 17, after a severe paroxysm of coughing, the right lung suddenly filled with bile and mucus; coughing and expectoration with occasional efforts to vomit continued constantly for two hours, as much as 2 ounces (59.147 cc.) being ejected at one effort; 24 ounces (709.7 cc.) of bile and mucus were ejected during two hours. The hiccups continued. Friction was heard anteriorly over the chest. On May 18, there was little cough and no bile in the expectoration. On May 19, the patient was comfortable, coughing very little; the mucus expectorated was occasionally slightly tinged with bile. There were occasional hiccups. Congestion of the lungs occurred several times during the day. On May 20, the patient was much better. He did not cough. In the afternoon, distressing hiccups developed that lasted all night. On May 21, after a paroyxysm of coughing, a sudden gush of bile rushed into lung. There had not been any bile since May 17. A pain developed in the shoulder and the hiccups ceased when bile was coughed up. On May 22, there was no cough or expectoration. On May 23, the patient had a slight pain in the right shoulder. At 3 o'clock in the afternoon after violent attack of coughing, the lung filled with bile; almost a pint of pure bile was coughed up and vomited. The cough and expectoration continued all night. On May 24, the patient was in collapse; he was stimulated; he coughed a little but did not expectorate. On May 25, the patient was free from cough and expectoration. On May 26, pain developed in the right shoulder; a paroyxysm of coughing occurred between 11 and 12; more than a pint of bile was coughed up from lung. The cough and expectoration ceased at 5. On May 27, and for several days before, an occasional quantity of healthy pus with matter was expectorated. On May 28, there was an effusion of fluid in the chest. On June 4, the patient's condition improved. On June 21, there was a dulness in the lower part of the right lung posteriorly and a slight uneasiness in the side. There is no record of the case beyond this point, and the ultimate outcome is not known.

Fouché's Case[54] (Gomet's Patient).—A girl, aged 20, was admitted to the hospital on May 18, 1885, with a history of intermittent fever at the age of 2 years; and a light attack of variola at 10 which left no traces. She had always been

delicate, and had been admitted to the hospital twice during the last year for a syphilitic complaint. On May 17, toward 11 p. m., she suddenly became ill without any previous symptoms with violent pain in the right hypochrondrium which kept her awake. She tried to get up in the morning, but had to return to bed. She vomited bile all day and night and came to the hospital the next day. During the examination, she held her hand over the right side, and pain radiated to her right shoulder. She was nauseated, but did not vomit; she had loss of appetite, but no jaundice. The stools were a gray, lead color and almost liquid. The urine was frankly icteric; chemical tests were positive for bile. The liver was not enlarged. From May 18 to 25, the condition remained the same, except that her temperature rose to 38 C. (100.4 F.) and she suffered from distention. Dyspnea began on May 26, and increased. The diagnosis was diaphragmatic pleurisy. On May 28, the phenomena of perihepatitis and peritonitis had almost disappeared, but pain persisted in the region of the liver and above it. Increasing difficulty in respiration was noted; the percussion note was dull, the breath sounds diminished, and fine râles were heard over the right lung in the back. The dry cough came in bouts. On May 29, there were more sharply defined signs of pleurisy. On May 30, after a bout of coughing in the night, a frothy yellow liquid, which looked like bile, was expectorated. The expectoration contained bile, but no trace of pus; microscopically, it showed fat droplets, pus cells, and bilirubin crystals, but no elastic fibers. The expectoration was pure bile. On June 1, the expectoration continued, being more abundant by day than by night. The patient was dyspneic, only a trace of bile was found in the urine. On June 2, there was no bile in the urine. On June 5, the expectoration increased to 700 cc. From June 6 to 9, the expectoration diminished to nothing; the cough was rare and dry. On June 10, she felt increasing oppression, and had a sudden bout of coughing with filling up of the lungs. The sputum became thicker and less yellow. A new analysis showed a diminution of bile and an absence of pus. Aspiration was performed without result. On June 11, the patient had a good night, without cough or expectoration. On June 15, she was well and she was discharged from the hospital on June 30.

GASTON'S CASE.[87]—A middle-aged man had severe symptoms of impacted biliary calculi, including absence of bile from the stools, jaundice, itching and tenderness over a fixed point a little to the right and below the ensiform cartilage. There was also peculiar and persistent pain in the right arm. There was sudden improvment in the patient's condition, with a change in the character of the stool, which contained a number of gallstones. The size of some of these gallstones caused the author to suspect that there was an ulcerated communication with the bowel. The patient became convalescent and went from the United States to Brazil. A few weeks later, he came home again; he expectorated an offensive bile stained sputum, and died within a few days after reaching the United States. Postmortem examination showed, ulcerations connecting the gallbladder with the duodenum, and an opening through the diaphragm into the lungs which were adherent to the diaphragm. The common duct was completely closed: its walls were thick and hardened. The collection of disorganized bile and serous exudate, which doubtless was originally confined in the dilated sac of the gallbladder, had made its way into a cavity formed between the upper surface of the liver and the lower surface of the diaphragm and extended through the aperture into the pulmonary structure. The flow of this offensive matter into the bronchial tubes seemed to be the immediate cause of death.

GRAHAM'S CASE 1.[6]—A man, aged 33, had had but one illness, which occurred three years before his case was reported. He was seized with pain in the region of

the gallbladder accompanied by fever, but not by jaundice. The pain radiated to the back. He was in bed for three weeks, and the attack lasted for six weeks, but he complained of pain in the hepatic region both before and after attacks. He recovered completely and remained well until the commencement of the present illness. On Nov. 27, 1886, he was seized with coughing and pain in the right hypochondrium, 3 inches (7.6 cm.) to the right of the median line and a little below the costal margin. Nausea and vomiting also occurred. The vomitus was a greenish-yellow transparent substance and continued for five days till December 2. At that time the pulse rate was 120; the skin was hot and dry. He had a severe chill the night before. On December 3, examination of the chest showed an area of hepatic dulness much increased, and pleuritic friction was heard immediately below the right nipple. The pain here was severe. The patient had an intense headache, the pulse rate was 140, the temperature, 104. He was vomiting freely. The bowels were constipated and the urine was highly

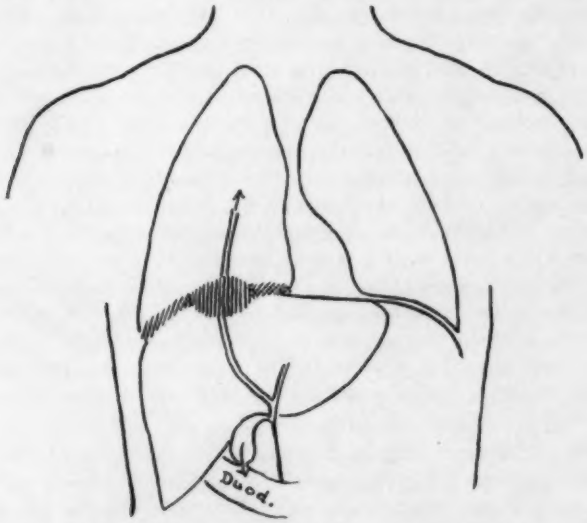

Fig. 8 (Gaston's case).—Subphrenic abscess with a break through into the bronchus; obliteration of the common duct; spontaneous cholecystenterostomy.

colored. During the evening, he began to cough up bile. On December 4, he coughed up bile freely, and complained of a burning sensation in the right side. He had to cough several times before the bile came up, and the bile was dark green. The temperature was 102 F.; and the pulse rate was 100. The pain became less severe and the headache less intense. The condition remained the same until about December 9. At that time, the pulse rate was 108; the temperature 102 and there was slight jaundice. He coughed up bile at short intervals, having two or three attacks of coughing during the hour. He raised a large tablespoonful of bile at each bout of coughing, the bile came only when he coughed, and it amounted to a half a cup full a day. On examination, it was found to be pure bile. The urine also contained a small quantity of bile. The deep liver dulness was on a line below the right nipple in the sixth interspace. A friction sound was heard over the upper surface of the liver anteriorly. Coarse mucous râles were heard over the anterior region of the right side of the chest; the posterior region was normal. The patient gradually improved, the amount

of bile being less daily, until it suddenly ceased on December 19. He was back to work Jan. 1, 1887. During the attack, the bowels were constipated, the feces were a light grayish color. He remained well until April 13, when he began to cough while riding in a street car. He had a choking sensation and expectorated bile. There was no pain. After four or five days, he was back at work. On April 21, examination was normal. From April, 1887, for over ten years, he was in good health, except for alternating attacks of constipation and diarrhea. During the latter part of this time, he noticed light colored stools and itching. In early May, 1896, he noticed that he began to be jaundiced. On May 31, he felt a sudden severe pain in the region of the gallbladder, which gradually increased, and soon after the onset, he coughed up a greenish fluid. He remained in bed for one month. At that time the urine was dark red. The attack passed away in two weeks. He continued to cough up bile from May 31 to November 22, with the exception of two or three days when he expectorated a large amount of mucus. The amount of bile varied from a few ounces to 2 pints. The skin was not normal in color, but it was not remarkably jaundiced. When the expectoration ceased, the jaundice deepened. On Nov. 20, 1896, he showed slight distention. A distinct swelling that was more or less tender and somewhat elastic was noticed below the costal margin. He complained of pain in the region of the gallbladder. The spells of coughing were more distressing when he was in a recumbent position. The sputum contained bile and mucus but no pus. Vocal fremitus was slightly increased on the right side. Dulness was elicited on percussion from 1 to 2 inches (2.5 to 5 cm.) higher than normal between the midaxilla and the sternum. Various râles were heard in the same area, also friction. There were small amounts of bile in the urine. During the cough, the patient was cyanosed. The temperature varied from subnormal to normal, but was never elevated. In December and January, 1897, he again coughed up bile, the amount varying from ½ to 2 pints and it was mixed with frothy mucus. Gmelin's test was positive. The patient's general health declined during the summer, and he was much weaker in December. On Jan. 26, 1897, an operation was performed. From 6 to 7 ounces (157.4 to 187 cc.) of dark bile were aspirated from the gallbladder; the common and hepatic ducts were full of stones. The ducts were over one-half inch (1.27 cm.) in diameter. A cholecystenterostomy was performed, the hepatic flexure of the colon being used. The patient died on the twelfth day from hemorrhage. He coughed up less bile after the sixth postoperative day. The cough tore open the suture line on the third day. On the sixth day, he coughed up material suggesting fecal contents, but it was negative for *B. coli*. The temperature rose. There was a consolidation in the lower part of the anterior right lung and the patient suffered from hemoptysis. The sputum contained blood and bile. On the ninth day, the blood increased. Death occurred on the twelfth day. A postmortem examination was not performed.

Graham's Case 2.[6]—A man, aged 25, was in excellent health up to the time of an accident. On January 28, he was kicked by a horse over the cartilages of the fifth, sixth, seventh and eighth ribs. He was not knocked down and there was no marked contusion or injury to the cartilages. He immediately felt a severe pain beneath the part struck and in the right shoulder. He walked to the house and the pain increased. Four hours later, he suffered intense spasmodic pain in the hepatic region and in the shoulder. The abdomen was much distended. There were frequent eructations of gas, which relieved the pain. He expectorated mucus tinged with blood. The pain was relieved by morphine for four days, then pneumonia developed in the lower anterior part of the lung; the inflammation subsided in about a week, but the consolidation remained. Fluid formed in the

right pleural cavity. Eighteen days after the accident, he began to cough up bile in small amounts after severe attacks of coughing and later he coughed up larger amounts with less effort, but never more than a pint in twenty-four hours; as a rule, less. He expectorated mucus and bile. On the twenty-sixth day after the accident, the right pleural cavity was aspirated, 1½ pints of bile stained with serum and pus being obtained. An opening was made on the following day and a tube drain was inserted. The bile drained for about a week, then seropurulent drainage was maintained for a longer time. The expectoration of bile increased after the operation; jaundice did not occur at any time. After operation the temperature fell to normal, but it rose again after a week. Coincidently, an increased area of dulness was observed in the hepatic region. One pint of bile and mucus was aspirated. A second operation was performed on the thirty-third day after the accident, portions of the sixth and seventh ribs being removed; a tube was inserted and mucus and bile were drained. After this operation, the stools became colorless for the first time. Five weeks later the patient was pale, emaciated and could sit up with difficulty; pulmonary resonance was heard only to the ninth interspace in the right side of the chest posteriorly; anterior superficial resonance was normal to the fourth rib; then there was absolute dulness to the sixth rib; below the sixth rib to the costal margin a tympanitic note was present. Two drainage tubes were inserted; when these were removed and the patient coughed, the quantity flowing through both openings was increased, and bubbles of air or gas produced foam. The amount of gas which passed through the lower opening was much greater than that which passed through the upper. The openings did not communicate (fluid put in one did not come out the other). At various times during the progress, the bile flowed in different channels; through the bronchi, through the pleural cavity and out through the upper opening, into the cavity on the convex surface of the liver and through the lower opening and through the common bile duct. The patient suffered severe pain in the right shoulder after the second operation, when the finger was introduced into lower opening. The flow of bile increased after ¼ grain (0.015 Gm.) of mild mercuric chloride was given every hour for five or six doses. Two and a half weeks later, the patient was recovering slowly. Bile was found in the bowel. The patient coughed up little bilious fluid, and little found its way out through the lower opening.

De Havilland Hall's Case.[10]—In 1876, a patient complained of anginal symptoms. He had been in good health until 1881, when he suffered from pain in the region of the liver, followed by jaundice. Other attacks followed with increase in the jaundice. Ultimately he vomited bright colored bile and large quantities of bile stained fluid, subsequently ascertained to come from the lungs. The patient made a good recovery, so that the anatomic lesion remained obscure.

Heaton's Case.[11]—A middle-aged woman had been ill for three months with chronic hepatitis, and complete dulness over the right side of the chest was found later. The right side of the chest measured 2 inches more in circumference than the left. She had intense pain, relieved only by large doses of morphine. Suddenly she expectorated a large quantity of pus, and in the course of six hours, nearly a chamber full of matter was said to have been expectorated. When this large discharge ceased, the expectoration, which continued, was tinged with bile. Great relief from the pain followed the escape of pus, but she was much prostrated. When she entered the infirmary, the more marked symptoms had subsided; there was no fever, the pulse was feeble and there were deficient resonance and obscure respiration in the right side of the chest and pain under the axilla. She did not remain long in the infirmary, and her further progress is not recorded.

Ido and Yasuda's Case.[1]—A woman, aged 20, who was admitted to the hospital on April 20, 1910, had suddenly had a boring pain in the epigastrium one night; it did not radiate, and continued for a week. At the age of 17, she had suffered from general weakness and malaise and icteric urine without demonstrable cause; a week later, she had had icterus. In the course of the disease, symptoms of pneumonia had developed suddenly. A bitter tasting sputum varying in color from that of an egg yolk to a greenish color was expectorated in large amounts. After three weeks the patient had recovered. Since that time, attacks of coughing would occur, after a rise in temperature, and the patient would expectorate gall-like sputum. Attacks of from five to fourteen days' duration recurred many times yearly.

Physical examination revealed an infantile person, backward in development; poorly nourished, and slightly anemic. The epigastrium was slightly sensitive to pressure; dulness of the liver in the right mammary line began at the fourth rib,

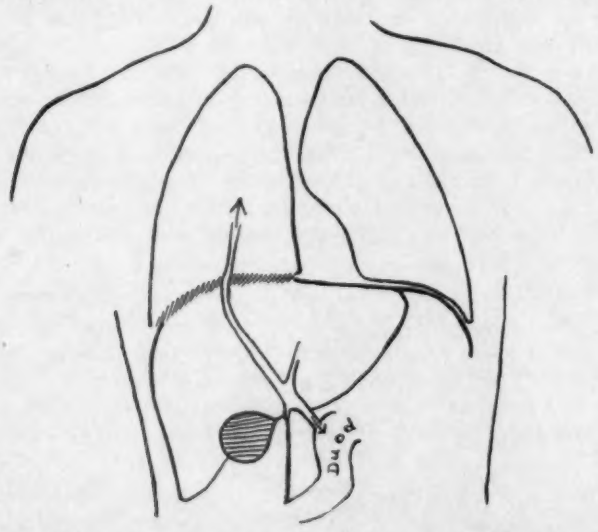

Fig. 9 (Ido and Yasuda's case).—Empyema of the gallbladder and fibrous pericholangeitis; spontaneous fistula of bile ducts to duodenum and fistulous opening through the diaphragm to the bronchi.

the lower border being hard and sharply defined, and three fingerbreadths below the costal margin. The surface of the liver was uneven, knobby and coarsely granular. The spleen was somewhat hard and enlarged. The feces were pale and foul. Parasites were not found. There was a slightly dull area anteriorly and posteriorly at the base of the right lung, and there were signs of bronchitis in the remaining parts of the lungs. Sputum was expectorated in small amounts; at this time, it was colorless, foamy and slimy, showing microscopic pus cells, lung epithelium and various micro-organisms. Results of examination for tuberculosis bacilli were negative. There were: Hemoglobin, 62 per cent; red blood cells, 2,670,000, and white blood cells, 10,000. The Wassermann reaction was positive. The urine was icteric. There was a distinct reaction to Gmelin's test, and there were traces of urobilin and albumin. A roentgenogram showed a diffuse broad shadow in the lower part of the right lung. On the third day, the temperature was from 38.9 to 39.4 C.; a dull pain was felt in the right side of the

thorax, and during the day the patient coughed and expectorated from 100 to 150 cc. of yellow stained, bitter-tasting, tenacious, slimy, in part seropurulent sputum. Stratification did not occur. Microscopic examination revealed numerous pus cells, few eosinophils and lung epithelium and probable liver cells, but no parasites. The feces were clay colored and foul. Icterus was increased markedly. On the next day the sputum was greenish. On the fifth day, the sputum was deep green, from 200 to 250 cc. being expectorated; the cough and bronchitis increased. On the sixth day, the sputum was yellow again, the quantity being somewhat diminished. On the ninth day, the sputum was colorless and a minimum quantity was expectorated. These attacks recurred four times in entirely analagous sequence. The patient was admitted to the hospital on April 29, 1910; on July 19, she suffered with pain in the abdomen, loss of appetite, repeated chills, fever and marked diarrhea. On July 21, she died, after a profuse vomiting of blood. A diagnosis of syphilis of the liver and a fistula between a bronchus and a bile duct was made. Autopsy revealed: The right lung entirely adherent to the wall of the chest and the diaphragm, the lower median part of the lower lobe scarcely being separable from the diaphragm; obliteration of the common and cystic duct; chronic empyema of the gallbladder; fibrous pericholangeitis; dilatation of the hepatic ducts and the bronchus; fistula of the hepatic duct to the duodenum, 1.5 cm. below the pyloric ring, and fistula of the hepatic duct to the right lateral bronchus. The bile passage was dilated a fingerbreadth, and had markedly thickened and fibrosed walls. The passage led through to the right side of the liver lappet in a straight direction down into the main branches of the hepatic duct and communicated indirectly with the hepatic fistula to the duodenum. There were universal icterus and anemia.

Kehr's Case[25] (according to Oliani).—Symptoms of bronchobiliary fistula disappeared little by little. When laparatomy was performed a shriveled gallbladder was found which communicated with the stomach at the pylorus. According to the author, the bronchial tree fistula healed when this communication formed. It is felt that this is a case of spontaneous healing of a pulmonary fistula through the formation of a fistula between the gallbladder and the pylorus.

Klauber's Case.[26]—A woman, aged 36, had suffered with intense cramps at varying intervals since she was 18, and for twenty-three weeks had been very sick. During this time, she had a severe attack of gallstone colic with pain under both costal margins, radiating to the back; she vomited after meals. The gallbladder was palpable and the liver increased in size. Since the second day of her illness, she had had severe icterus, chills and a temperature as high as 41 C. (105.8 F.), which subsided after a week, but she still had colic of diminished intensity. The stools were white at first; later they were colored, but a brighter color than normal. The urine was blood red with a brown foam. A careful search did not disclose any stones. There was a family history of tuberculosis. The patient coughed on entering the hospital and fourteen days before entry there was dulness in the right side of the back. On Sept. 23, 1906, she coughed up blood. On September 24, a great amount of purulent fetid sputum was expectorated which contained many leukocytes but no tubercle bacilli. This purulent sputum continued until the morning of September 27, when the patient coughed up a green, bitter-tasting fluid. She coughed up during transportation to the hospital a jar full of bile. This was brought up with slight effort. It amounted to a quarter of a liter, and was a foamy, green, thick, ropy fluid which reacted to Gmelin's bile test. The patient could lie still scarcely a few moments because of the tormenting cough. She was sleepless at night and exhausted by the cough. The lower half

of the edge of the liver was sensitive on pressure. The dulness of the liver was lower than normal, the upper border being in the seventh interspace. Few râles were heard over the dull tympanitic zone. On September 28, operation was performed; the liver showed venous hyperemia. The gallbladder was large and the entire region was bound down with wide dense adhesions. The gallbladder was opened and emptied of 50 cc. of dark green clear bile and soft stone (this could be mashed up with the fingers to a paste). Other stones were not palpable. A fistula could not be found. The gallbladder was drained. The condition ran its course without fever or complications. After waking, the patient coughed up a minimal amount of biliary fluid. From the afternoon of operation, the cough which raised bile ceased, and bile did not flow from the drain. Eight days after operation, the stool was colored. Only mucus came from the drain. On November 3, the patient was well. She left the hospital and went to work. Two

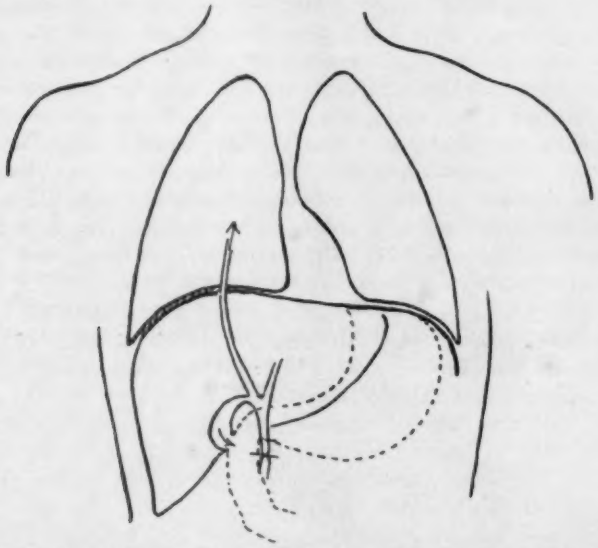

Fig. 10 (Kehr's case).—Spontaneous healing of pulmonary fistula by cholecystenterostomy.

years later (October, 1908), she again had pain in the epigastrium, vomited, was jaundiced and had acholic stools. The expectoration was stained a slight yellow once (the patient suffered from chronic bronchitis), but bile was never coughed up again. On Feb. 2, 1909, an operation was performed. There were adhesions to the intestines and the liver adhered to the abdominal wall. The gallbladder was the size of a hen's egg. There was a tear on the medial side of the former operative site, from which a white, thick, odorless fluid emptied. Suddenly, a white bladder appeared in the operative field. Echinococcus was found in the liver and adhesions separated in the region of the hepatic duct from the liver; a hard crackling paper-like mass was palpable, manifestly a calcified echinococcic wall. The cyst cavity was many chambered and very deep. Both the mother and daughter cysts were expressed. Two drains were inserted. On the following day, bile flowed from the cyst in the liver cavity. The stools were acholic for five weeks. The jaundice cleared up and the wound healed slowly. Another incision was made posteriorly under the twelfth rib. The fistula closed on May 24, and the patient was discharged on June 5.

KORTE'S CASE 5A[42] (according to Eichler).—A patient, aged 31, had had recurring lumbago; at one time it was accompanied by icterus. On one occasion, she had a cleancut attack of pain in the region of the gallbladder. Three weeks previously, she suddenly coughed up yellow, bitter-tasting pus. On admission, the patient was slightly jaundiced. Over the right lung, there were indefinite diminished breath sounds and sparse râles. Bile stained pus was expectorated abundantly. The diagnosis was abscess of the liver, perforating into the lung, probably caused by gallstones. After operation by the transpleural incision, the wound healed.

LABOULBÈNE'S CASE.[23]—A man, aged 46, was admitted to the hospital on March 14, 1875. He always had had good health; had not had syphilis, jaundice or hepatic colic. On Oct. 4, 1874, while in perfect health, he took a journey on a train. During the journey, he drank a large amount of beer. On the next day, coughing without great effort, he expelled a green sputum; at the same time, he commenced to notice a disagreeable bitter taste. His tongue was yellow. He had never vomited pus, liquid or membranes. Since October 4, five months in all, the green expectoration had continued, but had never been fetid. His appetite had not diminished. The stools were normal. Coughing prevented him from sleeping at night, and when he did sleep a little, he had bad dreams like those of an alcoholic person. According to the patient, the expectoration was more abundant some time after a meal. Examination revealed a healthy appearing man. Disseminated râles were barely heard in both lungs. Palpation and percussion disclosed hypertrophy of the liver. The sputum was not foamy but was greenish yellow without a foul odor and contained a few bits of unstained mucus. When one listened carefully, one could hear numerous mucus bubbling râles in the middle half of the base of the right lung. Percussion in this part gave a little more obscure note than did the opposite side. While being examined, the patient had a fit of coughing, and expectorated some biliary sputum without great effort. At the same time, the râles almost disappeared. His condition did not change until April 24; then the sputum became small in amount, and there was less bile. Microscopically, there were granular rounded nuclei with yellow pigmentations and a large number of leukocytes. Bloody fluid was present in other parts of the field and very small clots, very rare blood cells and some epithelium of the mouth. There was a gradual diminution in the amount of sputum until no more was raised. The signs at the base of the lungs cleared. The liver was diminished in size. A later note says that the patient had actually been healed.

LEBERT'S CASE[13] (according to Courvoisier and Oliani).—A girl, aged 15, suddenly became ill with chills, high fever, diarrhea, abdominal pain and great prostration. After five weeks, symptoms of pneumonia developed in the lower part of the right lung, followed by pneumothorax. Biliary sputum was expectorated for three months. Death resulted. Postmortem examination revealed numerous ascaris worms in the three large bile passages, and one macerated ascaris in the liver, and an abscess communicating with the bile passages. A large subphrenic abscess had broken through the diaphragm into the right pleura. pyopneumothorax occurred. There were sievelike perforations into several bronchi.

LUZATTO'S CASE[40] (according to Oliani).—A woman, aged 58, two years previously had had irregular fever and an intense cough for one month, after which she had expectorated a bitter green-yellow sputum. She became progressively emaciated. Pain developed in the epigastrium and in the hepatic region; death resulted. Autopsy showed a pulmonary cavity in the apex of the right lung. The

lung was adherent in toto to the ribs and to the diaphragm, in which there was an opening of 22 mm. communicating with the hepatic parenchyma and with the pulmonary tissue; there was a cavity with calcified walls with infiltration of biliary pigment. The duration of the condition was twenty-one months.

MACDONALD'S CASE.[23]—A butcher, aged 49, was admitted to the hospital on April 12, 1890. He had had syphilis in 1864, and in 1872 had lost his right eye and the whole soft palate through syphilitic ulceration. He had had malarial fever five months previously. One month before admission, he had had a dry cough which lasted one week. At the end of that time he had coughed up a large amount of purulent material. For two weeks this type of expectoration had continued, and he had easily raised large amounts when he leaned forward. The sputum had become thicker and less in amount and a few days before admission it had become dark. Physical examination revealed a poorly nourished man. Submucous crepitant râles were heard over the whole of the right lung, but were more marked toward the base; râles were heard also over the posterior lobe of the left lung. A constant irritable cough developed. The liver was palpable to within 1 inch (2.5 cm.) of the umbilicus. The expectoration was frothy and brownish. On April 20, a large amount of mucus heavily charged with bile was expectorated. The cough continued to be especially violent at night. The expectoration stained his moustache, beard and hankerchief a bright yellow. Bile was absent from the stools. On April 27, the expectoration increased in amount. The conjunctivae had an icteric tint. The urine was the color of porter. Slight anasarca occurred in the right foot. On May 1, the biliary sputum had increased, an almost incredible amount of bile thus being expectorated. Most of it was raised from one half to one hour after eating food. The cough kept the man and the other patients awake. The stools were free from bile. On May 20, all expectoration ceased and none was raised for four days, but after that it appeared in increased amounts. On May 30, the râles had cleared up. The dulness of the liver had diminished. The stools were still white. He was put on antisyphilitic treatment because it was believed that the obstruction was due to a syphilitic lesion which caused either occlusion of the hepatic duct by ulceration or more probably a gummatous growth pressing on it. On June 7, he was apparently improved. The cough and biliary expectoration was less. On June 19, the cough and biliary expectoration had ceased. Bile now appeared in the stools for the first time since April 20. On July 14, he was discharged. The abdomen showed a slight amount of ascites. The condition of the lungs was satisfactory. On July 15, he presented himself again. He had a violent attack of coughing during which he expectorated three small biliary calculi, two of which were facetted and about the size of a split pea; the other about one third of an inch long and one eighth of an inch in diameter forming a perfect coil of a tube (bronchial ?). The cough ceased after the stones were coughed up. The patient has since returned to work feeling well, and has not suffered pain or colic of any kind.

MANDARD'S CASE.[33]—A man, aged 70, became sick in November, 1849. For some time, he had suffered with vague and irregular pains at the base of the chest, until he was seized with fever, dyspnea, cough and violent pain in the right thoracic region. A diagnosis of pneumonia at the base was made. The disease did not follow the regular course, and instead of terminating by resolution, passed to a chronic stage. There was abundant expectoration of purulent sputum, which was thought to be due to an abscess of the lung, but the sputum soon took on other characters. It became brown green, then bilious. At the same time, the amount of expectoration increased definitely, with fits of coughing which brought up quantities of bile. On auscultation, there were all the signs of a cavity at

the base of the lung. The patient became increasingly weaker, and he died in the first part of April, 1850. Autopsy revealed extreme wasting of a normal sized liver with an abscess the size of a thimble, which occupied its anterior edge. There were old peritoneal bands in the region of the liver. The gallbladder was fixed to the diaphragm along the entire extent of its base. It was larger than normal, and the walls were thickened. When opened, it communicated with the thoracic cavity by a rounded opening. The base of the right lung was completely adherent to the diaphragm; the two upper lobes of the lung were healthy; in the lower lobe, there was a cavity having the volume of two walnuts; it was irregular and tortuous and had indurated walls which communicated below with the gallbladder and the apex of which was continuous with a large bronchus by means of distinct opening. In the interior of the sac formed by the gallbladder and this cavity there were numerous calculi—some in the gallbladder, others in the lung—which was bathed in a mixture formed of bile and pus. At the lowest

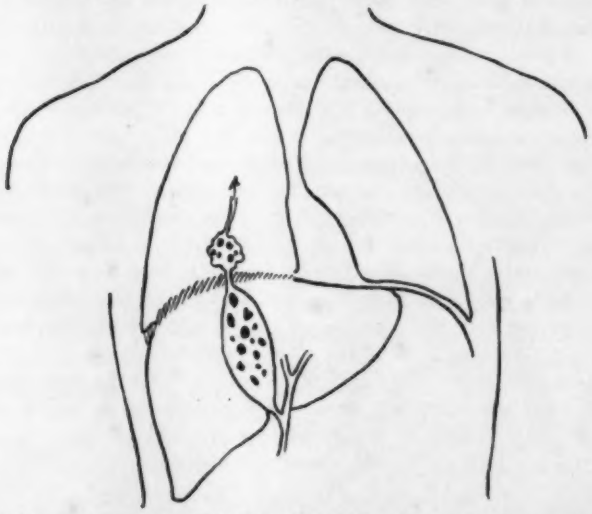

Fig. 11 (Manard's case).—Dilated gallbladder full of stones adherent to the diaphragm communicating across the diaphragm with a cavity in the lung which also contains gallstones; this in turn communicates with the bronchus, hour glass type.

part there was a type of pus (mud) formed by a large amount of fine gravel, which would have filled a coffee spoon. The canals were intact.

Mayo-Robson's Case.[43]—A man, aged 28, was well until April, 1894, when he had an attack of pneumonia. Six months later, he had an illness accompanied by cough, and on the third day, he began to expectorate. Shortly afterward, he coughed up a large quantity of pus and bile. He had not had any liver symptoms before this attack, except on one occasion when he had pain in the region of the gallbladder. Since that time, he had regularly coughed up bile and pus for a period of nine years. He was thought to have had phthisis, because of the rapid loss of flesh and night sweats. In May, 1903, he was round shouldered and old looking, and his fingers showed marked clubbing. The chest showed diminished breath sounds on the right side to the level of the seventh rib. The liver was decidedly enlarged, but not tender, 3 inches below the costal margin. He was slightly jaundiced. Bile was absent from the feces but it was present in the

urine. From 1 to 1½ pints of extremely offensive bile and pus was coughed up in twenty-four hours. The pus was crowded with bacteria. There were no tubercle bacilli or elastic fibers, and manifestly the pus was not caused by a bronchiectasis. On May 21, 1903, operation was performed. The liver was enlarged and cirrhotic. There were firm adhesions from an old shrunken gall-bladder and the lower surface of the liver to the stomach, duodenum and colon. A gallstone the size of a small nut was found impacted in the hepatic duct. It was removed. Numerous adhesions were found between the dome of the liver and the diaphragm. The hepatic duct was drained. The expectoration of bile was immediately arrested, but pus continued. The expectoration of bile returned on the third day, and then gradually became less. The cough gradually diminished, and the purulent expectoration rapidly decreased. The wound healed in a month. Two months later, the patient was much improved, and was gaining weight

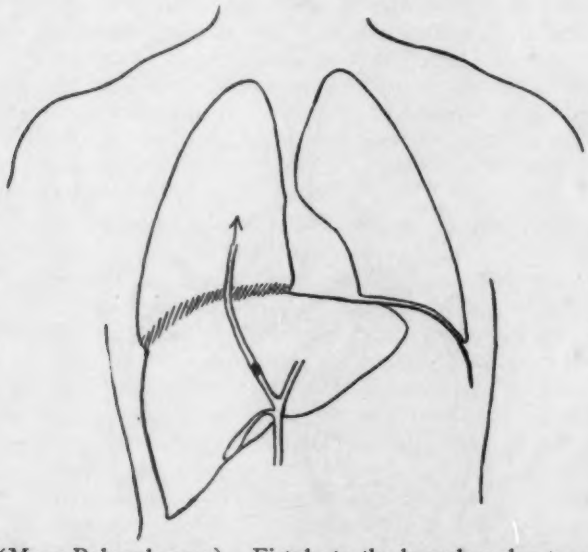

Fig. 12 (Mayo-Robson's case).—Fistula to the bronchus due to a stone in the right hepatic duct.

rapidly, coughing up only small quantities of mucopus without bile. There was no jaundice, all bile passing by the bowel.

NERMORD'S CASE.[21]—A woman, aged 50, was admitted to the hospital on April 13, 1891. She had had scarlatina at 10 and angina at 17. She was always well until 1883, when she suddenly had a chill and vomited bile (about a basin full). Immediately after that, continuous pain developed, which was localized to the right hypochondrium. It was so intense that she could not bear the weight of even a sheet in that region. In the evening it was noticed that she had become yellow all over and that the stools were white. The pain lasted four or five days, but disappeared following the use of purgatives; then the jaundice lessened and the stools became colored. She remained weak for three or four weeks, her appetite returning little by little. Every year for eight years, she had two or three days of acute pain in the right hypochondrium, accompanied by a subicteric tint of the skin and decoloration of the feces, but this condition cleared up promptly and she resumed work. In October, 1890, there was renewed pain, but it was less than usual; jaundice occurred, and the stools soon became

decolorized. She retained her appetite, but became thinner. From October to March 13, at 3 p. m., she had a chill and attempted to vomit; acute pain developed drium. She continued to work, but daily grew weaker and thinner. On March 13, at 3 p. m., she had a chill and attempted to vomit; acute pain developed in the hepatic region. The stools became decolorized. She had become increasingly jaundiced for two or three days. On March 14, she was better; the pain had almost disappeared, and her color was commencing to become pale. On March 21, the abdomen, thighs and feet were swelling. She remained in bed until March 30. On April 13, she was very weak, and ascites increased daily. She entered the hospital. Examination showed a very thin jaundiced woman with sunken eyes and a feeble voice. Ascites and dilated veins were present on both sides of the abdomen. The border of the liver was felt one fingerbreadth below the costal margin. During the day and night of April 16, she expectorated frothy, slightly yellowish mucus. On April 18, she coughed up about a basin

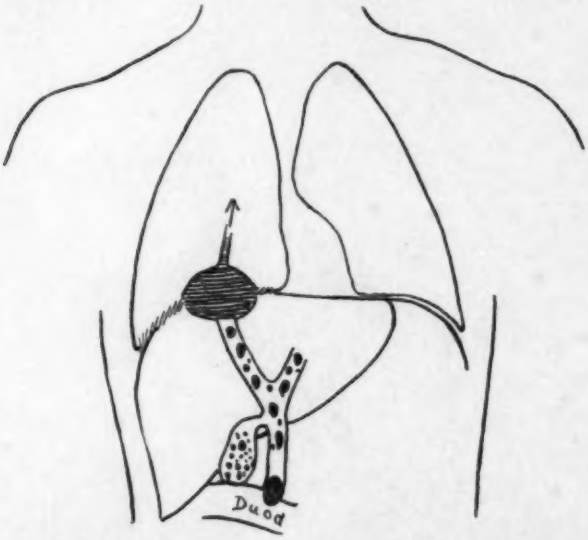

Fig. 13 (Nemord's case).—Subphrenic abscess with pulmonary fistula; the bile duct and the gallbladder crammed full of stones.

full (1,200 Gm.) of greenish, slightly thick liquid with yellow foam; she was sure that it was not vomitus. The stool was without color; there were bile pigments in the urine. Puncture of the liver in the convex border showed only blood. On April 19, she coughed almost all day and night, raising 1,000 Gm. of the same type of sputum. Her mouth was dry and there was a bitter taste. There were mucous râles in the lower two thirds of the right lung. On April 20, she had a stubborn cough during the night; thick, green masses of sputum mixed with mucus and some blood, 700 Gm., being expectorated. On April 21, she coughed continuously, but especially in bouts, 300 Gm. of sputum being expectorated. On April 23, 300 Gm. were expectorated. On April 24, 300 Gm.; on April 25, 250 Gm., and on April 26, 250 Gm. The bouts of coughing were less frequent and not so severe. On April 27, she coughed little and there was no expectoration. The abdomen was tapped, 9 liters of slightly greenish non-albuminous liquid being removed. On April 30, the patient passed a bad night. She had fits of coughing, expectorating 200 Gm. of liquid bile. On May 2, a

thick dark green bile on which floated some nummular material was coughed up. The patient was very tired. On May 3, 500 Gm. was expectorated. On May 5, the sputum was so thick that she was scarcely able to expectorate it. Acute pain had developed in the right hypochondrium the day before, radiating to the right shoulder. She was very much oppressed, and continued to lose strength; the least movement caused fatigue. A puncture was performed near the right kidney and a slightly bile stained liquid was removed. Death occurred on May 6 at 10 a. m. All the time she was in the hospital the feces were without color.

Autopsy revealed an enlarged liver, with a slightly irreguar, granular, green colored surface. The extra hepatic and intrahepatic bile channels were greatly enlarged and crammed with biliary calculi. The common duct was the size of the small intestine. There was a large calculus at the intestinal orifice. The gall-bladder was adherent to the duodenum by its fundus. It contained biliary calculi and little bile. On cutting the liver, the bile channels were dilated and filled with calculi in considerable quantities as if a solid had been injected and then fragmented. They were bathed in a purulent greenish liquid. The liver, diaphragm and base of the right lung were adherent at the posterior costodiaphragmatic sinus. Here there was a cavity the size of mandarin with irregular walls flattened from above. It contained a green liquid mixed with pus and bile. The fistulous tract on the biliary side could not be found, but on the pulmonary side it was traced through to the bronchus.

OETIKER'S CASE.[25]—A man, aged 46, was admitted to the hospital on Sept. 10, 1925. His health had been good until one year previously, when he became ill with icterus and an enlarged liver. After a rest, he felt fairly well and worked until three months before he was admitted to the hospital. At this time icterus again appeared and disappeared together with a sudden onset of a cough with the expectoration of bile. There was no pain, but striking emaciation and weakness. Examination shows sclerae with a subicteric tint, otherwise there were no signs of jaundice. The liver was firm and somewhat enlarged. It was painful, and there was a nodular tumor of the left lobe reaching almost to the umbilicus; it was only slightly displaced on respiration. An area of dulness like a second heart figure mounted over the dulness of the liver two fingerbreadths to the right of the sternum. He had emphysema of the lung, bronchitis and pleurisy in an area the breadth of the hand in the right posterior inferior part of the chest. There was a daily expectoration of bile mixed with mucus varying in quantity from 800 to 1,100 cc. The appetite was good. There was no vomiting, and the stools were acholic. The temperature was normal in the morning; in the evening it rose to 37.8 C. (100 F.) and exceptionally to 38.4 C. (101.1 F.). In a roentgenogram, the upper edge of the liver was indistinct and passed over into a cloudy consolidation of the right lower lobe. When iodized oil 40 per cent was injected, the bronchi of the lower lobe appeared widened with irregular inlets, especially in the thickened part. Further examination after four days did not show any iodized oil in the liver. The sputum did not contain any tumor cells, scolices or hooklets. Eosinophilia did not occur. The Weinberg and intracutaneous tests with echinococcus fluid were negative. A differential diagnosis was made between abscess, tuberculosis, echinococcus and malignant disease of the liver with a breaking through and encroachment on the lung. It was decided a definite diagnosis could not be made. The condition was probably multilocular echinococcus or carcinoma.

The patient asked urgently for operative relief because of the extraordinarily troublesome cough which gave him no rest at night. When the abdomen was

opened, the subserosa was very edematous. Weblike loose adhesions occupied the entire gallbladder, duodenum and region of the diaphragm. After separating the adhesions, a thick mass in the right lobe of the liver and adherent to the diaphragm was palpable. The growth was posterolateral to the summit of the liver, and appeared either as a tumor or a multilocular echinococcus. Retraction of the liver appeared to be too slight for the latter. The liver was somewhat enlarged and showed a round edge. Puncture did not reveal any pus, only cells; at a second puncture brown fluid was found. The examination of the stomach was incomplete on account of adhesions. The region of the pylorus was free but about the head of the pancreas there was a thick, slightly movable resistant mass, which appeared to be a primary cancer. It was not certain that this was the pancreas itself. Cholecystenterostomy was performed. Death occurred the evening after operation from heart failure.

Postmortem examination revealed cancer arising from the head of the pancreas and completely involving the papilla of Vater in the duodenum. The common

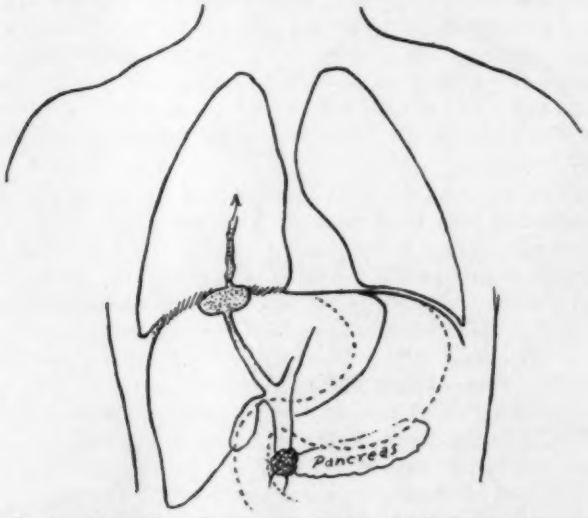

Fig. 14 (Oetiker's case).—Cancer of the head of the pancreas with a block of the common duct; tuberculosis at the dome of the liver with a fistula extending through into the lung and bronchi.

duct, cystic duct and intrahepatic bile passages were markedly widened. A conglomerate tubercle, the size of a mandarin was found in the liver close to the convex surface. It was hard, showing cheesy decomposition in only one small area. The liver was adherent at this point to the tuberculous diseased diaphragm and the lung. The conglomerate tubercle communicated with the bile passages. The tubercle bedecked fistula led directly from the bile ducts into a widened bronchus. In the lungs there was also a purulent bronchitis, bronchiectasis, old pleural adhesions on the right and a diffuse, thick crop of miliary tubercles somewhat larger than in the lower lobe of the right lung. In the upper lobe of the right lung a small calcified mass was found. Isolated miliary tubercles were found in the liver. The bronchial and mesenteric glands were tuberculous and calcified, the latter group being markedly affected. Cancer metastases were not found. The cancer caused bile stasis, flare-up of tuberculosis, breaking through to the lung. Fresh tubercles in the lung and liver were due to a gland breaking into a vein.

PARSON'S CASE.[64]—A young man was seen on February 2, with a temperature of 103 F.; he had a pulse rate of 128 and a very dark skin. Extreme tenderness was elicited in the right hypogastrium with considerable swelling and dulness extending above the costal cartilage. Deep percussion revealed the left border of the liver 5 inches (12.7 cm.) to the left of the median line. The gallbladder was also greatly distended. Expectant treatment was instituted. The condition remained unchanged for five days. The stools were white and the urine contained bile. On February 7, a dry hard cough developed and on February 8, he began to expectorate large quantities of bile, shown by nitric acid and Pettenkoffer's test. He became delirious and sank rapidly. On February 10, the sputum became normal in color. An enema gave a healthy dark passage from the bowels. Convalescence was rapid and on February 14, he was about the house, though he was much emaciated.

PERRIER'S CASE.[65]—A man, aged 34, was admitted to the hospital on April 30, 1900. He gave a negative history as to his present illness. Eleven years previously he had noticed a sharp pain in the region of the liver on the right side. Some days later, a frank hepatic colic developed. One month later, he had another attack which lasted eight days. Every year since, he had had similar attacks during the month of December. During the interval, he was well and did not have any symptoms. He was said to have had pleurisy on the right side five years before admission. He remained at rest for a month. The symptoms were slight so he did not remain in bed. In March, 1899, his color turned yellow some days after he had noticed that his urine had a dark tint. The jaundice increased until the end of June. He was weak, but did not suffer. Pigmentation then diminished rapidly, but he felt ill at ease and tired and was without forcefulness or courage. A tumor appeared in the epigastrium, and the right side of the hypochondrium was enlarged. The pain became severe, immobilizing him completely. He vomited often. That winter the tumor diminished soon after the appearance of a troublesome cough, accompanied by expectoration of bile. The cough was almost continuous for six weeks. He spat up almost continuously small mouthfuls of a greenish foamy, often very bitter liquid. Sleep was impossible on account of the asphyxia. He could sleep a little with his head supported against the bed. He suffered from considerable weakness and emaciation. The liver and the spleen were enlarged; over the lower fifth of the right lung posteriorly there was a zone of tympany; the percussion note was flat. Breath sounds were diminished. There were moist and cavernous râles. The biliary expectoration did not contain pus or false membranes. He had never observed anything but bile with the exception of a slight amount of blood for one or two days. The urine and feces were normal. Operation was performed, the eleventh and twelfth ribs being removed. Adhesions were not present. The pleura was opened deliberately to create pneumothorax and to cause collapse of the lung. Intense dyspnea was felt in the evening. The expectoration diminished. A violent pain developed in the side; the temperature was 38.5 C. (101.3 F.) and profuse sweats developed. There was progressive amelioration of the symptoms on May 11. On the following day signs of general bronchitis appeared. The biliary expectoration increased progressively until it became as abundant as before operation. The wound suppurated. Blood was found in the sputum occasionally. Since the evening of June 24, blood mixed with bile had been expectorated in considerable quantities. In the morning, the expectoration was almost exclusively biliary. On July 4, he left for the country. Every day during the last days of the report he had a true hemoptysis. The

64. Parson: Boston M. and S. J. **106**:190, 1882.

temperature was always between 37.7 and 39 C. (99.8 and 102.2 F.). A further record of this case was not given.

Roper's Case.[42]—A woman, aged 56, had always suffered with bilious attacks. In September, 1910, she had had typical gallstone colic, and one year before a slight attack of jaundice. Before this she had had a similar, but less severe, attack of pain and had vomited and had slight jaundice. In September, 1911, there was no bile in the stools. A burning of the hands and the feet was experienced. Under treatment, she gained 5 pounds (2.3 Kg.) and bile reappeared in the stools. In February. 1912, she had an attack of pleurisy on the right side. In the middle of March, 1912, the feces were pale. The symptoms in the chest were diagnosed diaphragmatic pleurisy. A rub was heard in the anterior part of the axillary space; the temperature was 103. Any motion was painful, and she could only lie flat on her back. On March 16, she began to expectorate bile in the

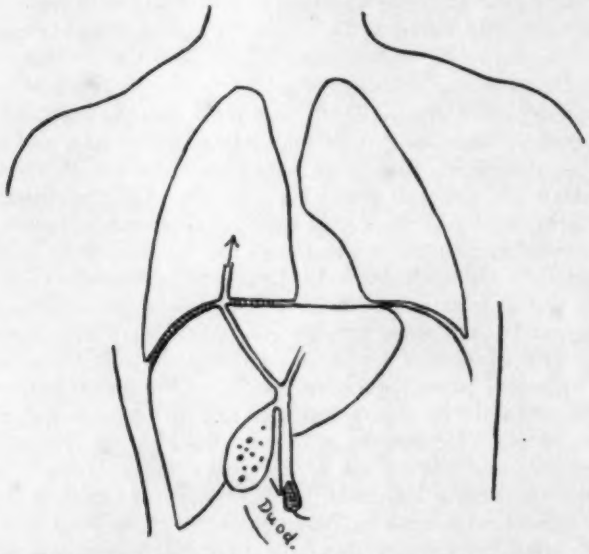

Fig. 15 (Roper's case).—Fistula in the bronchus with a stone in the common duct and stones in the gallbladder.

course of an ordinary cough. On April 5, very faint icterus was present. She coughed slightly every few minutes. She expectorated about 2 teaspoonfuls of golden bile occasionally accompanied by a little mucus. She sat erect in bed, because she thus reduced her cough to a minimum; lying back caused incessant coughing. The cough was slight and suggested rather the welling up of fluid into a bronchus which at a given level caused a short, soft clearing cough totally different from the violent straining of bronchitis. Twenty ounces of almost pure bile was raised in twenty-four hours; sucking in of lower right intercostal spaces occurred; she did not have dyspnea, and the percussion note was unaltered. An insufficient amount of air entered the lower lobe of the right lung; there were marked expiratory sound and coarse râles; vocal fremitus and vocal resonance were absent. The liver was plainly palpable 2 inches below the ribs; it was hard, but not tender. The urine and the feces did not contain bile. On April 9, an operation was performed. A large distended gallbladder with some small stones was found; a hard mass in the common duct near the duodenum, measuring

1 inch in diameter and about 2 inches in length, partly extruded in the duodenum. The common duct was incised and a mass spooned out; it was cheesy, gritty, "too soft to evacuate as a whole," and weighed 2 drachms. The gallbladder was incised, and the stones were removed. The patient coughed once on being put to bed, spitting up a pellet of black bile-stained mucus. Toward morning, on April 10, about a teacupful of frothy mucopus stained a dirty brown was coughed up. The cough was troublesome, and there was some bronchitis. The patient's progress was uneventful. She was out on the sixteenth day. The sinus discharged bile for six weeks. Generally there was no expectoration during the day, but every morning she coughed for a gradually decreasing time and emptied some cavity in her lung; the amount evacuated was about a teacupful for the first few weeks, and then it lessened gradually. On June 12, 1912, the sinus had healed; the product of expectoration was reduced to about six specimens of nummular sputum each morning. Further record of the case was not made.

SCHLESINGER'S CASE.[41]—A man, aged 64, entered the hospital on Jan. 27, 1906. He had been healthy until 1904, then for two weeks, he had fever and severe pain in the right costal margin and shoulder and he vomited greenish fluid. Jaundice was not present. Two months later, he again had pain in the region of the liver, loss of appetite, constipation and hiccups. The attack lasted longer than a month. The urine was dark. On the right side, there was a massive dulness at the apex toward the axilla and a dulness forward on the base of the lung but not posteriorly. Friction rub could be heard over the liver. Breath sounds were diminished at the base in the area of dulness, and vocal fremitus was diminished. The skin over the dull area was edematous. Jaundice was not present. There was no bile in the urine, but urobilin was demonstrated. A puncture in the sixth interspace in the midaxillary line gave a yellow, clear exudate, containing polymorphonuclears. The patient was discharged after five weeks. From that time he coughed continuously and had pain on pressure over the margin of the right ribs. After two months, without any special previous pain, he suddenly coughed up so great a quantity of greenish colored, clear fluid that was not foul as nearly to suffocate him. This measured about half a liter. Since this time, the expectoration had been greenish and had a bitter taste. In spite of great effort, only a little sputum was raised. During the month before he was admitted to the hospital, the patient was markedly emaciated and often had fever. He was constipated.

Examination showed an emaciated man without jaundice or edema. His breathing was labored and accelerated. There was a slight even rise in temperature. Outspoken acra cyanosis was noted. The right side of the thorax lagged. The percussion note at the apex and the base of the right lung was shorter, higher and somewhat tympanitic. Dulness was present from the mamillary line axillaward in a straight line, limited by the upper edge of the sixth rib, and extended somewhat toward the scapular line. Bronchial breathing was heard in the dull area; directly over the dull area, there were numerous, medium and fine vesicular râles. Over the remaining part of the lung, especially at the right apex, isolated coarse râles were heard. Vocal fremitus was absent in the dull zone; bronchophony could not be elicited. The edge of the liver was not palpable; percussion over the liver and the dull area of the thorax was painful. A tumor was not found in the region of the liver nor was there any free fluid in the abdomen. The urine was normal. The sputum was moderately abundant, mucoid, ropy and stained a deep green-yellow; it had a stale odor and an alkaline reaction. In unstained preparation, much mucus and fatty degeneration epithelial cells from the mouth were found but no neoplasm, scolices, hematoidin crystals or liver cells. Elastic tissue and other tissue were not demonstrated, and few leukocytes. There

was only a small amount of protein. A marked bile reaction occurred. A stained preparation showed a few polymorphonuclears and fewer eosinophils. There were numerous small diplococci. *Diplococcus lanceolatus* (extracellular); rare short chains of small streptococci and *Micrococci catarrhalis* were found. *B. coli, B. tuberculosis,* or *Pseudomonas aeruginosa* was not present, otherwise the flora was that of the cavity of the mouth. Culture revealed streptococci and *Diplococcus lanceolatus.* Three days later, the man died from heart failure.

Postmortem examination revealed adhesions of the omentum to the colon and about the right lobe of the liver. A thick-walled passage 5 cm. long and 2.5 cm. wide on its inner circumference was found in the region of the gallbladder; it was lined with mucous membrane. Posteriorly, this passage widened somewhat. On the upper surface of the passage toward the liver, a blunt-edged gallstone the size of a pea was found in an ulcer. From this the cystic duct ran backward, 4 cm. long, narrow and hard-walled. It opened into a markedly

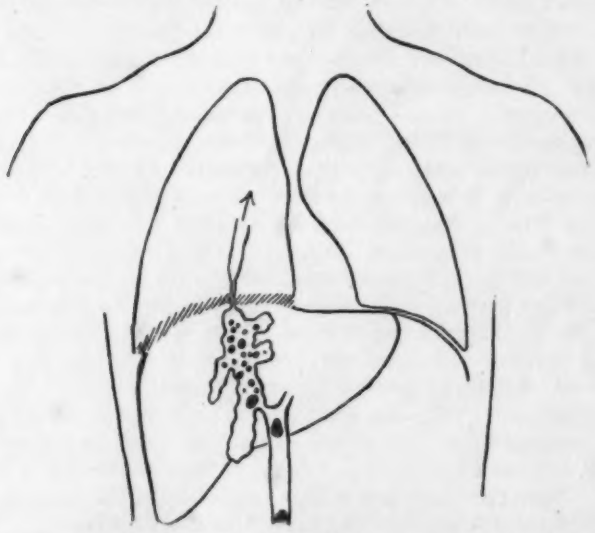

Fig. 16 (Schlesinger's case).—Gallbladder connected with large cavity in the liver, full of stones; stones in lower end of the common duct, fistula through the diaphragm into the bronchus.

widened common duct 3.5 cm. in circumference, in which lay an oval gallstone the size of a hazelnut with rough surface, which could be pulverized between the fingers. On the upper surface of the liver, which was firmly adherent everywhere to the diaphragm, was a many-bayed, ramifying cavity with broader communications with the remnant of the gallbladder, with distinct, thick walls and a rather smooth inner surface. It contained over twenty blunt-edged, facetted gallstones varying in size from that of a pea to that of a bean. From this cavity a sharply circumscribed passage, the size of a pencil, led through the diaphragm and ran between this and the fast adherent lung posteriorly. It communicated with a bronchus the size of a goose quill, which led into the main right bronchus. The lung tissue in the region was pasty. The right lung contained numerous ramifying distinct cavities at the apex, with viscous shredded walls of scaly thickened tissue. The entire right lung was interspersed with numerous small, in places confluent, white-yellow, caseous patches at the base, usually with cavities in their center.

SCHULTZE'S CASE.[36]—A woman, aged 25, was admitted to the hospital on Feb. 6, 1873. She was generally healthy, except for minor ailments. From the middle of August, 1872, for ten weeks there had been icterus with marked pain in the region of the liver. In December, 1872, the condition had recurred, but after six weeks in the hospital she had returned to work. On Feb. 3, 1873, icterus had developed. On the evening of February 5, she had had a chill followed by fever, severe headache and pain in the right hypochondrium, nausea and vomiting. Examination revealed marked icterus. The patient was doubled up with intense pain in the region of the liver and the right shoulder. The temperature was 40.7 C. (105.2 F.); the pulse rate, 108. Percussion in the region of the liver was scarcely tolerated. On February 7, the fever was 41 C. (105.8 F.). On February 8, she had herpes labialis, and the liver was enlarged. On February 10, the liver extended from the fourth right rib to the navel. Icterus was present until February 13, and then a clear reaction was not demonstrated in the urine.

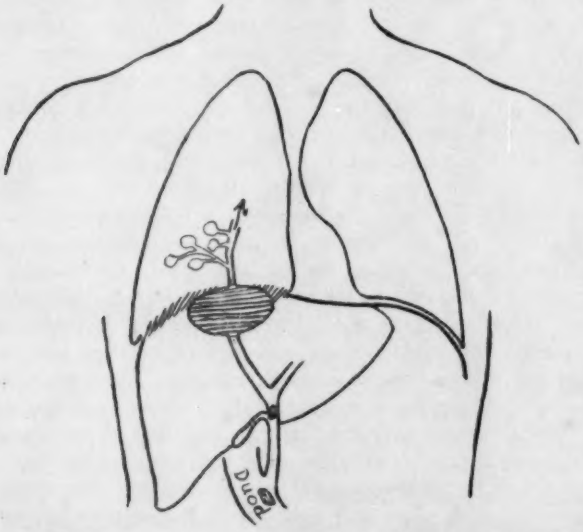

Fig. 17 (Schultze's case).—Subphrenic abscess with fistula to broncheietic cavities at the base of the lung. Stone at the junction of the cystic and common ducts.

The fever remained high after the diminution of icterus. The stools remained remarkably gray to yellow gray. Gallstones were not found. Coughing was not marked. On March 8, a scanty, slimy material mixed with a slight quantity of blood was coughed up. The lungs were normal On March 25, after she had been nauseated for a day, had vomited once and had had a feeling of marked oppression, a large quantity of stale-smelling, but not fetid sputum was expectorated. The sputum was the color of the yolk of an egg and was coughed up in abundant quantity at various intervals. Microscopically, the yellow color was not scattered in a diffuse way through the whole mass of the sputum, but was present as smaller or larger clear yellow flecks in individual preparations. These yellow flecks disappeared when potassium hydroxide were added, and the entire preparation no longer had a uniformly yellow color. Addition of nitric acid changed the yellow to green. Partly destroyed pus cells and a great quantity of hematoidin crystals composed the great mass of sputum. There was expectoration of this

material on March 28 and 29 and on April 1 and 2, after which the sputum lost its yellow egg-yolk color, became gray-yellow, then gray-white, and finally had the usual mucopurulent character of the sputum in chronic pneumonia. For about two weeks, the quantity varied from 500 to 600 cc. daily, and stood in three layers, the middle layer being a thin fluid with masses. Hematoidin crystals were found for one week, but almost ten weeks after the onset, there were still fragments of crystals; later, these were not present. With the onset of the cough the fever began to drop. The tumefaction of the right hypochrondrium diminished and left a depression under the right costal margin; signs of a cavity were demonstrated in the lower part of the right side of the thorax, anteriorly. The patient looked wasted during the summer of 1873. The stools became lighter and lighter, and in August were often paper white. The urine and the skin showed only a trace of icterus. Progressive inanition was noted. By the end of June, there was intermittent fever to 40 C. (104 F.) every two or three days. In August, the fever diminished. In September, fever and intense icterus were noted. There was bile in the urine, and the skin was yellow. A tumor the size of a walnut appeared in the region of the gallbladder and disappeared after six days. There was color in the stool the day the tumor disappeared; the next day the stool was normal and they remained so from that time. The cough increased in intensity in September and the patient had fever, the temperature becoming as high as 40 C. (104 F.). In October and November, the bronchitis increased in severity and the patient became weak. Death occurred on Dec. 23, 1873, after she had been in the hospital eleven months. The diagnosis was gallstones, abscess of the liver and a bronchial fistula (direct or through a subphrenic abscess), and pneumonia following diffuse bronchitis. Postmortem examination revealed the left lung adherent to the pleura; there were peribronchial and pneumonic patches. The right lung was entirely adherent; the tissue, especially of the lower posterior and middle parts was full of smaller and larger cavities communicating with dilated bronchi in many instances. There were fresh bronchopneumonic patches. The anterior surface of the liver was adherent to the wall of the abdomen, right transverse colon, right kidney and descending part of the duodenum. A wide cavity between the lung and the liver bordered on the ribs anteriorly and inferiorly; the upper surface of the liver was filled with a discolored fluid, mixed with slime and air. There was a smooth opening through the diaphragm to the bronchus in the lower lobe of the right lung. The cavity was larger than an apple; the right lobe of the liver was behind it, the dilated bile passage, below. A thin partition was found between the convex surface of the liver and the cavity in the liver, which in turn opened into the fistula. The gallbladder was very small. There was a fistulous opening from the common duct to a duodenal ulcer. The bile passages were widened. Two gallstones were found at the junction of the common duct, cystic duct and hepatic duct; one was the size of a hazelnut, the other, fragmented and the size of a pea.

SENDLER'S CASE.[9]—A woman, aged 48, was admitted to the hospital on April 30, 1906. She had had measles, scarlet fever, pneumonia, puerperal fever and inflammation of the cecum. For six years, she had had pain under the right costal margin associated with jaundice, vomiting and fever, often severe. After two months, the attacks of pain recurred more severely, but they were accompanied by only slight jaundice. Eight days before admission to the hospital she had had a severe attack of vomiting of bile. Examination showed a fat, jaundiced woman, with a somewhat enlarged liver, but no tumor. On May 6, her temperature was 38 C. (100.4 F.). There was increasing pain in the region of the gallbladder. On May 8, operation was performed. From thirty to forty stones varying in size up

to that of a hazelnut were found in the gallbladder. It was removed and nine black, pea-sized stones were found in the common duct. Two days postoperatively, bronchitis developed. The stitches cut through during the attacks of coughing. On May 17, the temperature was falling, and the wound suppurated. On August 19, severe pain developed in the right side, the temperature in the evening was 39 C. (102.2 F.). Bronchitis developed in the lower lobe of the right lung, but was not striking, cough was present. The pain was localized under the right costal margin and radiated to the back; it occurred in attacks. On Jan. 4, 1907, the patient entered the hospital again, still complaining of pain in the right costal margin. She brought a letter from her local physician which stated that in the beginning of April, 1907, following much coughing and severe sticking pain, she coughed up a green, biliary, bitter sputum; otherwise, her complaints were as formerly. Operation was refused. After a little time, the expectoration became white again. The condition was better until a new attack which occurred in the middle of October, 1907. The patient still refused operation. Examination revealed weak breath sounds in the right side of the back, perhaps also in front on the right side, moist coarse râles, much coughing, abundant sputum, and a small but not painful liver. In November, 1907, she coughed up sputum that varied in color from yolk yellow to yellow-brown. Microscopically, there were few erythrocytes, many white blood cells, and mucus. On November 2, she had a septic temperature and coughed in attacks which lasted several hours unless morphine was given. She expectorated a large amount of yellow and green sputum, which gave clear bile reactions and tasted bitter. The urine contained some bile. Posteriorly, the right lung was dull to the axilla; large and middle sized râles were present. The liver was not increased in size; the stool was acholic. On November 6, there was less fever and the patient was subjectively better. There was less bile in the urine, and only a trace of it in the sputum. On November 8, there was slight icterus. Posteriorly, the lower part of the right lung was dull in an area measuring 5 or 6 cm. longitudinally; anteriorly, numerous sticky râles were heard, which became more audible higher up. On November 12, the temperature increased. The patient could not rest unless morphine was given. The sputum and the urine contained an increasing amount of bile. The patient was well nourished, but weak; and was annoyed by much coughing and a bile-stained, thick, purulent sputum. She suffered from severe pain in an area anterior to the axilla which extended between the eighth and eleventh ribs. Dulness was elicited over the same area and also over the lower lobe of the right lungs; bronchial breathing and râles were heard. On November 16, the seventh and eighth ribs were resected in the anterior axillary line. The lung was adherent in front to the axilla and the diaphragm, but was free posteriorly. The liver appeared to be adherent below; this was confirmed by incision of the liver. The cavity in the lung contained a foul-smelling, bile-stained abscess the size of a hen's egg, and a cavity about the size of a walnut was found in the liver. It emptied itself of a few yellow-brown tissue hulls resembling echinococci. In the depth of the cavity, there was a three-branched communication with a bronchus. A tampon was employed. Fever and jaundice developed the day after operation. The membrane was removed. Echinococcosis was diagnosed as microscopic examination revealed an echinococcic wall. On November 27, slow healing began; the expectoration became less, and was white. The wound of the previous operation was again opened widely on December 20. The bronchus opening was still easy to find, but no longer communicated with the liver. The cavity communicating with the bronchi was lined with epithelium. Energetic treatment was instituted with Paquelin's cautery; this was also applied to the surface of the lung

about the bronchi. On Jan. 10, 1908, the patient was recovering slowly. The sputum was slight. On March 11, the lung-lip fistula remained. On March 16, operation was performed; the scar and nearest rib were resected. The lung was freed from the diaphragm and wall of the thorax. On April 14, two small bronchi still were open. On August 8, the patient was discharged with the wound healed. On March 24, 1909, she reported that the wound had broken open on March 14, and had discharged a small amount of yellow secretion, but had again healed. The chest was in a satisfactory condition at the time this report was made, but the patient still complained of pain in the scar in the epigastrium. On June 22, the patient entered the hospital again with intermittent pain in the epigastrium; fever as high as 40 C. (104 F.), and icterus. The scar again opened in the back, discharging a yellow secretion. On July 11, a sudden sharp pain was felt in the epigastrium; there was slight icterus. An operation was performed and the old wound was opened. There were adhesions between the anterior surface of the stomach and under surface of the liver as well as between the upper surface of the liver and the diaphragm. A large facetted stone was found in the common duct behind the duodenum, then numerous small ones and finally broken stones. On August 2, the drain was removed. On September 21, erysipelas developed on the front of the wound and on the abdomen and shoulder. On October 11, a sudden sharp pain was felt in the abdomen and the patient was in a state of collapse. On October 17, the patient's condition was good; the appearance of the wound was satisfactory. After five weeks of fever, the wound healed. From November 23 to 28, the temperature was normal. From November 28 to December 10, there was a rise in temperature during the evening from 38.1 to 39.5 C. (100.5 to 103.1 F.); the wounds were diminishing and the fistula in the lung closed. Otherwise, the patient's condition was normal.

Smith and Rigby's Case.[40]—A woman, aged 50, was admitted to the hospital on Dec. 14, 1902. Eighteen months previously she had been in the hospital on account of acute symptoms of cholangeitis probably due to gall stones. Pain had developed in the right upper quadrant, associated with pyrexia and jaundice, but all the symptoms had subsided without operation. Since leaving the hospital, she had had attacks of abdominal pain from time to time, which lasted from one to two hours, usually after eating; both the onset and the end of these attacks were sudden. The skin was yellowish on several occasions, but never deeply jaundiced. For the last three months, she had suffered from pain in the back and in the right shoulder without relation to food; the pain was not severe. Ten days before admission she had had a severe fit of coughing accompanied by expectoration of green fluid with a very bitter taste. Since then a distressing cough and expectoration of similar fluid had persisted. Examination revealed a fairly well nourished person with a normal color. The conjunctivae were a little yellow. She complained greatly of cough, which was frequent and distressing. After each fit of coughing, she raised, with little effort, 1 or 2 drachms of dark green, frothy fluid expectoration with a bitter, unpleasant taste. She could not lie down owing to this constant desire to cough; she did not have a fever. The lungs were emphysematous and hyperresonant; expiration was prolonged, and rhonchi and râles were audible on both sides. The liver was palpated two fingerbreadths below the costal margin, superiorly it was not enlarged; there was slight tenderness on palpation over the region of the gallbladder; there was no rub. The sputum gave a characteristic reaction for bile. The urine and the stools were normal. For three weeks the condition remained unchanged. From 20 to 30 ounces of sputum was expectorated daily. On Jan. 5, 1903, the patient complained of increasing pain in the right hypochondriac region. On examination, it was found

that the local tenderness had increased. There was no change in the thorax. The patient remained the same until January 17, when the stools became pale, but not definitely clay-colored. The urine also showed a trace of bile. On January 21, the stools were clay-colored; the cough was the same, but increased when the patient lay down. When she lay on the left side, she coughed up more fluid. There was tenderness at the right costal margin. On February 4, there was no bile in the urine; the stools were only slightly paler than normal. The skin was slightly yellow, and the conjunctivae were definitely icteric. The amount of sputum was the same. On February 7, epigastric pain and clay-colored stools appeared. On February 17, operation was performed, and the adhesions of the gallbladder to the colon were released. The gallbladder was empty and contracted; the walls were thickened and fibrous. The cystic duct was slightly dilated; the common duct was the size of the forefinger. Calculi were found low down in the common duct behind the head of the pancreas. The common duct was opened and the two calculi were removed. Drainage was instituted through the gallbladder. The calculi were facetted, the larger one being the size of a marble, the smaller one, the size of a hazelnut. On February 18, she vomited bile-stained fluid and blood. The cough had almost ceased, so that the patient could lay on her back without much discomfort. She still coughed up mucus and bright green bile occasionally. On February 20, there was still a slight amount of bile-stained sputum. On February 21, her cough had almost entirely disappeared. There was little sputum, and it did not contain a trace of bile. By February 23, there was practically no cough. A fecal fistula opened. One month after the operation, she was sitting up; a slight amount of bile still discharged about the wound; the fecal fistula was closed. Later record showed that the wound healed entirely.

SMITTEN'S CASE.[10]—A woman, aged 23, complained of weakness, loss of appetite and cough, which became worse when lying on the right side. Illness had begun two and one half months previously with a cough. Three weeks before admission to the hospital her temperature had risen to 38 and 40 C. (100.4 and 104 F.), and remained high for eight days. A purulent-like fluid had been expelled on the third day; it had been yellow. On admission to the hospital, the temperature was 36.2 C. (97.1 F.). Percussion below the scapula on the right side was dull; the note was tympanitic around the area of dulness. The greatest tympanitic note was obtained between the scapula and the posterior axillary line. There was bronchial breathing here and above the scapula. A pleural rub was heard on the left side in front to the axillary line. The heart was two fingerbreadths to the left of the nipple line. The patient had to assume a sitting position night and day to save herself from choking. She could not lie on the right side. The author believed that she had a chronic subdiaphragmatic abscess which was responsible for the passage between the bronchus and one of the larger biliary ducts, and explained the total absence of bile in the intestine by inflammatory disease; he therefore decided to remove the obstruction to the passage of bile, taking the route through the abdomen. Under local anesthesia, an incision was made parallel to the costal margin. Normal bile ducts were found, but no adhesions. The incision was closed, and transpleural laparatomy was performed by resection of two ribs with exposure of the pleural cavity. The liver was held to the diaphragm by massive adhesions. The patient did not bear operation well because of cardiac trouble, and the procedure was discontinued. She died later. At autopsy the diaphragm was found cemented to the lower lobe of the right lung. A cavity, 5 by 5 cm. was found in the adhesions. A passage through the parenchyma of liver to one of the biliary passages was found, also a passage to one of the bronchi; the main bile duct was free from bile. There was diffuse bronchitis in the lung.

STUMPFF'S CASE[29] (according to Burgess).—A woman, aged 50, suffered from attacks of biliary colic from 1903 to 1907. She was operated on Dec. 19, 1907, and showed a perforation of the gallbladder with considerable infiltration along the common duct. Cholecystectomy was performed with gauze drainage. The wound discharged for two months, then healed. In June, 1912, she had diffuse bronchitis with a blood-tinged expectoration. Examination revealed the spleen and the liver enlarged and firm; albuminuria, 5 per cent. On August 24, the woman was knocked down by a bicycle. Three days later, she had severe pain in the right side and coughed up large quantities of thin, yellow, frothy mucus; the stools became colorless. Afterward, days of expectoration of bile alternated with days free from such expectoration. On September 16, pleurisy associated with effusion developed in the right side, but the fluid was free from bile. Eleven days later the expectoration of bile finally ceased. On November 14, death occurred after two days of peritonitis. Autopsy showed diffuse peritonitis, apparently extending

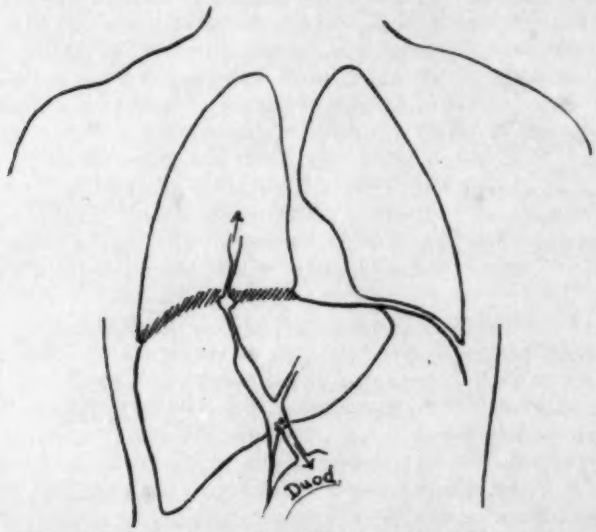

Fig. 18 (Stumpff's case).—Gallbladder has been removed; common duct obliterated by scar tissue; fistula to the duodenum from dilated ducts; stones still present in the biliary ducts.

from a phonephrosis; extensive adhesions of both the right lung and the right lobe of the liver to the diaphragm, and a small cavity the size of a pea at the posterior upper surface of the liver in the midst of the adhesions. This cavity contained bile-stained fluid and communicated both with a bronchus and with the bile ducts. The main bile ducts were much dilated. The lower end of the common duct was completely obliterated. A narrow fistula was present between the common duct and the duodenum just beyond the pylorus. Several small calculi and some bile sand were found in the common duct, the calculi being just sufficiently large to produce an intermittent complete blocking of the fistula.

TUCKWELL'S CASE.[30]—A woman, aged 60, was admitted to the hospital on Jan. 22, 1870. For six years, she had had severe attacks of vomiting, pain in the epigastrium and jaundice. Each attack lasted from three to six weeks. The last attack had occurred ten weeks before admission. The present one began the day of admission, with severe pain in the epigastrium and back, accompanied by vomiting.

Examination did not show anything remarkable. Two days later, she had a violent attack during the night accompanied by severe pain and retching. On January 25, there was a recurrence of the violent attack. Stones in the cystic duct was diagnosed. Her condition remained the same for three weeks; she vomited frequently and had pain between the shoulder blades. On February 18, severe chill and hepatic dulness, 1½ inches below the costal margin in the mammary line were noted. There was an area of tenderness on pressure over the right hypochondrium. On February 23, the patient had two attacks of chills and retched violently. On February 25, the pulse was feeble and the rate was 130. The pain in the back had become less severe and was localized to the right hypochondrium. On February 27, she had fits of coughing accompanied by expectoration of small purulent masses with a brownish tinge and a fetid smell. Resonance at the base of the right lung was impaired and friction sounds were heard on deep inspiration. On February 28, she coughed up a few ounces of brownish substance

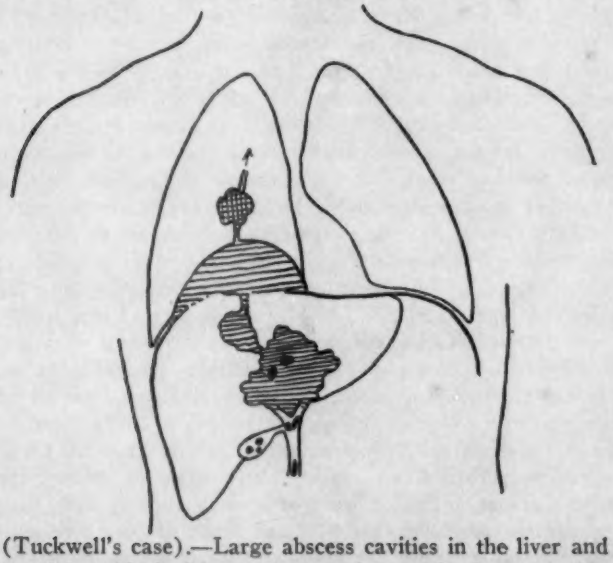

Fig. 19 (Tuckwell's case).—Large abscess cavities in the liver and subphrenic space; common duct blocked by stones; cavity on the lung; double hour glass type of fistula.

with a fetid odor. Her breath was fetid (gangrene lung?). Dulness increased on the right side, posteriorly, and bronchial breathing was heard. On March 2, she coughed up more foul-smelling material. Her condition was becoming progressively worse. The dulness now reached the scapula and sounds below were absent. She continued to bring up small quantities of sputum daily, never more than a few ounces of the same, foul-smelling brown expectoration, until March 4, when the eyes and skin were jaundiced for the first time. On March 6, the patient was deeply jaundiced. Death resulted. Postmortem examination revealed the gallbladder enlarged to twice its natural size. The right lung was compressed and pushed up by from 40 to 50 ounces of fetid pus in the pleura. One patch the size of a small apple in the lower lobe was gangrenous. The upper and posterior part of the liver was adherent to the diaphragm. The interior of the right lobe of the liver was converted into a large irregular cavity crossed by a partition of broken-down hepatic tissue surrounded by ragged walls. The cavity was filled with foul pus and contained several small biliary concretions and it

communicated below with the open-mouthed branches of the hepatic duct. The gallbladder was filled with dark bile and numerous gallstones of varying sizes. The common duct was dilated widely by a calculus the size of a small bean at the duodenal opening. The hepatic duct was blocked by a large dark concretion, which sent shoots into all the large terminal branches of the duct. All the bile ducts were much dilated. The fistulous opening was large enough to admit a small catherer. The opening led through the diaphragm from the pleura to a smaller suppurative cavity in the liver (an offshoot from the large abscess) and thence by a large open-mouthed branch of the hepatic duct into contact with one of the offshoots from the large concretion.

Tyrman's Case.[33]—A man, aged 22, was admitted to the hospital on Nov. 18, 1908. He had fired a shot from a small caliber service rifle toward his right breast after being scorned in love, and had been picked up on the street, given first aid and brought into the hospital one hour later. Examination revealed marked collapse, outspoken pallor, a state of shock and bloody sputum. There was a hole the size of two farthings in the right mammary line at the fifth rib; the surrounding area was stained black. The exit wound, the size of one farthing, was in the right scapular line between the ninth and tenth ribs; there was a sucking wound. Dulness was heard over the right thorax, and tympany to dulness at the right apex. The patient was treated for shock, and the wounds were packed. Bleeding from wounds continued for three or four days, and the patient's condition remained unchanged. On November 25, the dressings were soaked with bile. Nearly 1,200 Gm. of necrotic tissue cells, pus and fibrin were drained. The stool and the urine were normal. On November 27, dressings were applied three times a day; the output of bile lessening generally. On November 29, there was about 800 Gm. of secretion, demonstrably more after eating, most marked on sitting up. To the author's amazement, the patient coughed up a quantity, from about 80 to 100 Gm., of slimy green bile, mixed with pus cells and very bitter to the taste. In the sputum glass, there was a larger lower layer of greenish thin fluid and an upper layer of clear, foamy, whitish-yellow fluid. From November 30 to December 21, the bile from the wound diminished to about 600 Gm. daily; there was more when the patient was in an upright position and sitting than when he was lying down. About 50 Gm. of secretion was coughed up daily. On December 22, under chloroform anesthesia, the fifth and sixth ribs were resected for about 12 cm. toward the axillary line. Adhesions between the lung and the diaphragm were freed by blunt dissection. A resistant strand about the size of a finger with a broad base was separated by instruments. A large wedge-shaped cavity was found with thick connective tissue walls, tenting up the diaphragm two finger-breadths at the place of separation of the strand. Possibly part of this may have been due to the convexity of the liver. At the base of the strand and immediately adjacent to it, there was seepage of bile. On December 23, a slight amount of biliary sputum was expectorated; an irritating cough was present; only a slight seepage of bile occurred. The record made on January 14, was that the course of the illness was uneventful; the wound had closed.

Van Wijhe and Hammer's Case.[34]—A woman, aged 57, entered the hospital on Aug. 7, 1920, with a complaint of hemoptysis. For fourteen days she had been coughing and expectorating bile. Since November, 1919, she had remained continually sick with grip, but it had not prevented her from working. Examination showed a small woman with kyphosis and emphysema, but no icterus. There was residual pneumonia in the lower part of the right lung. The liver was somewhat enlarged, but not painful on pressure. In 1883 and 1885, she had been admitted to the hospital and had been shown at the Congress of Dutch Surgeons. She had

had pain in the back for seven years, the onset being about her twentieth year, in 1883, when she had a large liver and a bulging on the right side of the thorax. Puncture of the liver had released a clear fluid and echinococcic hooklets had been demonstrated. Within the next two years, she had had two attacks of sickness resembling attacks of acute cholangeitis. Echinococcic cysts were found in the stools; they were found once while the patient was in the hospital. The enlarged liver slowly returned to normal. In June, 1885, thrombosis of the right and left femoral veins had developed. The patient had not been seriously sick since 1885 and did not have pain in the back. On entry to the hospital, she was weak and thin; her temperature was subnormal with sharp rises. The dulness in lung gradually increased. Albumin was found in the urine but no bile pigments nor urobilin. There were no clay-colored stools nor blood. Several examinations of the sputum did not reveal tuberculosis nor echinococcus. The daily output of sputum was between 50 and 600 Gm.; it was markedly bile-colored and rarely did

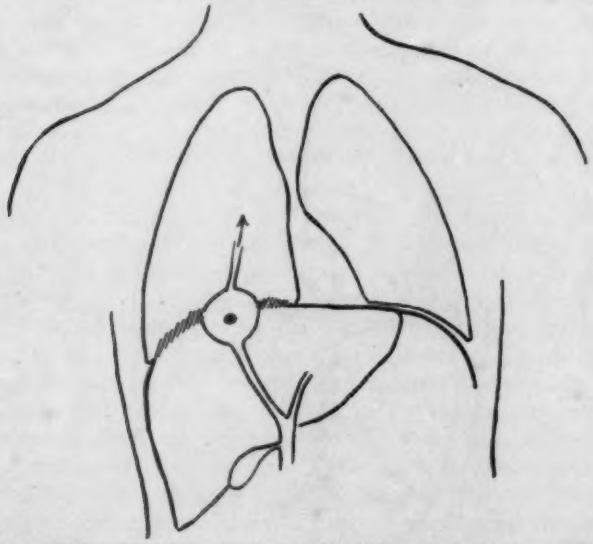

Fig. 20 (Van Wijhe and Hammer's case).—Subphrenic abscess with ball valve stone and fistula.

not contain bile. The complement-fixation test (Weinberg's method) was negative. A roentgenogram showed a "spongy" shadow in the right lobe of the liver as large as a Gülden. Because of weakness and dragging down by the expectoration of bile, it was decided to operate. On Sept. 19, 1921, the right lobe of the liver was exposed across the right pleural cavity. In separating the adhesions, a calculus was found in this region. This was partially removed and the cavity was tamponed with gauze. The patient did not survive the night. Postmortem examination revealed a cavity, part of which was in the liver and part above, lying somewhere to the right of the midline; bile passages connected with it and appearing like main branches of the hepatic duct; a fistula to the main bronchus of the lower lobe of the right lung and thick adhesions in the right side of the liver, the sternum and the right lung.

VISSERING'S CASE.[80]—A man, aged 63, was admitted to the hospital on July 19, 1895, complaining of pressure in the region of the stomach, loss of appetite and constipation for two days. The next evening, he had an intense chill and severe

pain in the abdomen, especially in the right lower quadrant, and repeated vomiting. He was bathed in perspiration and felt cold; the pulse was scarcely palpable, and the rate was rapid. There was an outspoken picture of threatened collapse and marked meteorism. Pain developed in the region of the cecum. Slight pressure here was painful. Considerable swelling of the liver was noted; the lower edge was two fingerbreadths below the costal margin. During the next week, the pain became localized more and more to the region of the liver. There was a marked swelling of the liver, one handbreadth below the costal margin. The patient became jaundiced and had clay-colored stools. The gallbladder was palpable; it was the size of a small apple and sensitive. The general condition was very unsatisfactory. On August 1, he had a chill with fever to 41 C. (105.8 F.), severe dyspnea; a picture of pleurisy of the right side and a painful cough with slimy sputum. Dulness was heard over an area, a handbreadth in width to right lateral and posterior part of the thorax. On August 10, the chest was

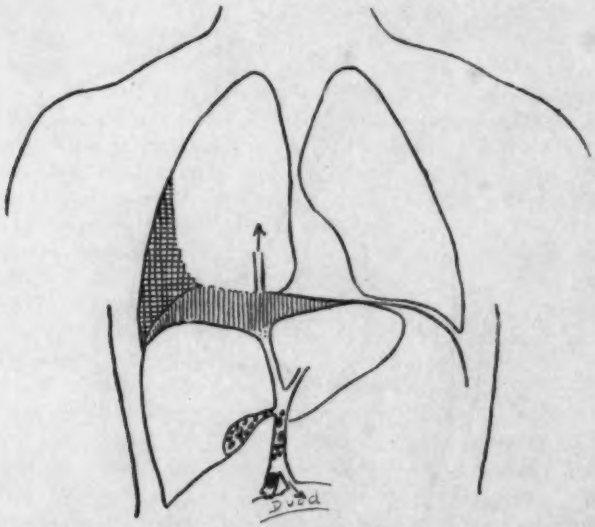

Fig. 21 (Weiler's case).—Subphrenic abscess, empyema, stone in the common duct and gallbladder; spontaneous fistula to duodenum and fistula to bronchi.

tapped, but no fluid was obtained; the patient refused a second puncture. With the onset of pleurisy and other symptoms, the swelling of the liver and gallbladder diminished. The gallbladder was no longer palpable. The pain disappeared. During September, he coughed up large quantities of foul sputum daily, which varied from pure pus in the course of next two days to egg-yolk stained sputum, and then to a strikingly thin fluid consistency. The patient complained of a bitter gall taste. The jaundice was reduced, and there were fewer clay-colored stools; there was a small amount of bile in the urine. On December 27, the patient coughed up gallstones. There was rapid improvement throughout January.

Weiler's Case.[60]—A woman, aged 45, entered the hospital on April 20, 1901. Twenty years previously she had had pleurisy on the left side. Ten years and six years previously she had had gallstone colic. She had been cured at Karlsbad. The patient said that her color had been very yellow, but that she did not know when the gallstones had passed. Ten days before admission, she became weak,

lost her appetite and had had fever. She had not had chills; but a burning and piercing pain had been present in right costal margin, in the back and in the right arm. The pain had increased. On April 19, the pain had been severe and continuous. Since then she had suffered from nausea and regurgitation of a bitter tasting, greenish, slimy material. She complained of shortness of breath and of a "sticking" pain in the right side. Examination revealed nothing remarkable except dulness in lower part of the right side of the chest. Puncture in the eighth and ninth interspace of the posterior axillary line was performed without result. On April 28, pleurisy developed in the right side. On May 9, a sudden sharp pain was felt in the right side and there was cough with expectoration; there was slight collapse. The sputum was slimy, uniform and abundant, with opaque, yellow yolklike specks. Microscopic examination revealed fatty degeneration pus cells, numerous bilirubin crystals and no elastic tissue. On May 10, the patient was pulseless, in a state of marked collapse and vomited. Death occurred at 7:30 p. m. Postmortem examination revealed a right-sided purulent pleurisy and compression of the lung. There was a subphrenic abscess in the right side of the liver. The base of the lung and surface of the liver adhered to the diaphragm. At the highest place in the diaphragm there was a perforation into the parenchyma of the lung. An old atrophic cholecystitis and cholelithiasis of the gallbladder, cystic and common duct were noted. A fistula of the common duct led to the region of the papilla duodenum. There was a duodenal polyp at this place.

YATES' CASE.[35]—A man, aged 47, in June, 1921, complained of loss of weight and a feeling of general weakness and illness. A few days later, a severe hiccup commenced, which lasted for nine days and then ceased abruptly. It was so severe that it deprived the patient of sleep and seriously interfered with eating and even with speaking. When it disappeared the only complaint was general weakness. Three weeks later, however, a cough began, accompanied by copious expectoration, which he described as being "like brown jelly and sometimes streaked with blood." This continued for one month, when the color of the sputum suddenly changed to yellow. It was now intensely irritating and associated with violent paroxysmal cough which sometimes lead to vomiting. He was admitted to the hospital on November 5. He had had scarlet fever at 8 and pneumonia at 16, and gonorrhea at 19. He had never been abroad, and had not had dysentery. He was coughing up large quantities of clear, bright yellow fluid which consisted almost entirely of bile and contained a few gram-positive cocci and one or two cells, but no pus. There were no traces of amebas or hooklets. He was emaciated and weighed 102¾ pounds (46.6 Kg.). Before his illness, his weight was 128 pounds (58.1 Kg.). He was not jaundiced. There was a small area of impaired breathing at the base of the right lung, over which medium crepitant râles could be heard. The breath sounds over this area were harsh and vesicular. Vocal fremitus and vocal resonance were not changed. The liver was a little enlarged, and it could be felt just below the costal margin, somewhat rounded and hard. There were several dilated veins in the abdominal wall chiefly on the right side above and below the umbilicus. Operation was performed on November 25, and portions of the eighth and ninth rib on the right side were resected; the upper surface of the diaphragm was explored. No opening was seen. The adherent surfaces were separated as completely as possible, and a drainage tube was inserted, from which a small amount of bile escaped for a few days. After operation, the coughing up of bile ceased completely for five days and then began again. The patient continued to raise a considerable quantity for some time, but it steadily decreased

and ceased altogether about a month after operation. He was discharged on Jan. 9, 1922, feeling well and free from cough, having gained several pounds in weight. The wound was healed. Crepitus at the right base of the lung had disappeared, but some dulness remained. Signs in the abdomen were unchanged. Six months later, he was well and doing full work. He regained his former weight. The enlarged veins of the abdominal wall had disappeared, and the only sign present in the right side of the chest was a slight persistent dulness.

MYOSITIS OSSIFICANS

A STUDY OF ANOTHER UNUSUAL CASE *

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My only excuse for reporting the following case of myositis ossificans is that failed to follow the usual course of this long recognized condition, and also because of several interesting points in the differential diagnosis.

In 1925, Adam Gruca [1] published a clinical and experimental study of this condition, with a detailed report of seventeen cases. He gives what I believe is the best classification to date. It is as follows:

1. Myositis Ossificans Traumatica:
   (*a*) Following a severe single injury by blunt force.
   (*b*) Following dislocations; i. e., elbow, clavicle, knee, shoulder.
   (*c*) Development of bone along the track of perforating gunshot wounds.
   (*d*) After clean incised wounds.
2. Myositis Ossificans Chronica:
   (*a*) After repeated slight trauma; i. e., "rider's bone," "soldier's shoulder."
   (*b*) Occupational.
   (*c*) Nontraumatic; history of trauma not obtainable.
3. Myositis Ossificans of Infectious Origin.
4. Myositis Ossificans—Para-Arthritica.
5. Myositis Ossificans—Neurotica.

In his article he also groups the numerous theories into two divisions: Those assuming that parosteal bone arises from the periosteum of the neighboring bone, and those attributing the condition to a metaplasia of local connective tissue.

Unfortunately, however, up to the present it has been impossible to prove these various theories experimentally, and until myositis ossificans can be experimentally produced, any case revealing new departures from the usual should be reported, as it is on clinical observations that one has to rely to solve the problem. Therefore, I am reporting the following case.

REPORT OF CASE

Present Illness.—E. S., an upholsterer, aged 28, was married and had two children. The family history was negative.

On March 9, 1923, while helping to lift an automobile from the mud, the patient felt a sharp, sudden pain in the upper portion of his right arm and shoulder. The following day his arm was tender, as was his shoulder and the muscles of the

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* From the Surgical Department of the Johns Hopkins University School of Medicine.

1. Gruca, A.: Myositis Ossificans Circumscripta, Ann. Surg. **82**:883, 1925.

neck on the right. A slight swelling without discoloration was noted, and hot applications were advised by his physician. As the swelling and slight pain did not improve, he was referred to me, April 7, 1923.

Physical Examination.—The patient was well developed and weighed about 140 pounds (63.5 Kg.); his physical examination was normal, except the right upper arm. A marked swelling of the outer side of the right upper arm, over the prominence and insertion of the deltoid muscle was present. The skin was normal in color over this area, and neither dilated nor enlarged veins were noted. The mass was markedly indurated, nearly bony in consistency, smooth and not lobulated

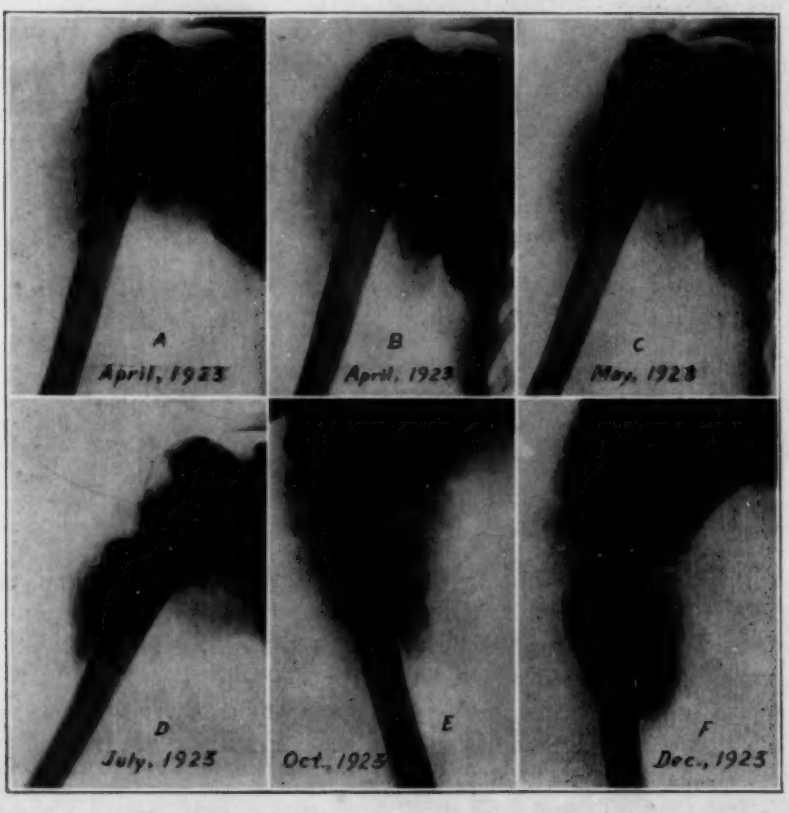

Fig. 1.—*A* and *B* present the roentgenographic appearance one month after injury, showing beginning bone formation in soft parts. *C*, *D*, *E* and *F* show the increasing bony formation.

and impressed one as being in the deltoid muscle; the skin was movable over the mass and the growth was slightly movable over the humerus. The mass was slightly painful on pressure and on full extension of the arm over the head. Motion of the shoulder, elbow, wrist, or fingers was not limited. The grip was normal. The radial pulse was the same as on the left side. Roentgenograms taken at this time (*A* and *B* in fig. 1) showed a definite bone-forming tumor of the outer side of the right humerus, which was thought to be due to the stripping up of periosteum at the insertion of the deltoid muscle, the result of indirect violence one month previously.

The patient was advised to stop work, apply hot applications to his shoulder, keep his arm in a sling and, notwithstanding a negative Wassermann reaction, he was given 45 grains (2.6 Gm.) of potassium iodide a day. The patient remained away from his work two months and then returned to his usual occupation because of economic reasons.

He reported to me again in October, 1923, on account of pain and increase in the size of the mass. At this time, seven months from the date of injury, the mass was larger, felt bony and seemed to be extending around the shaft of the humerus. It was firmly attached to the shaft along the outer side, and the tips of the examining fingers could dip under the mass on the posterior and inner side of the

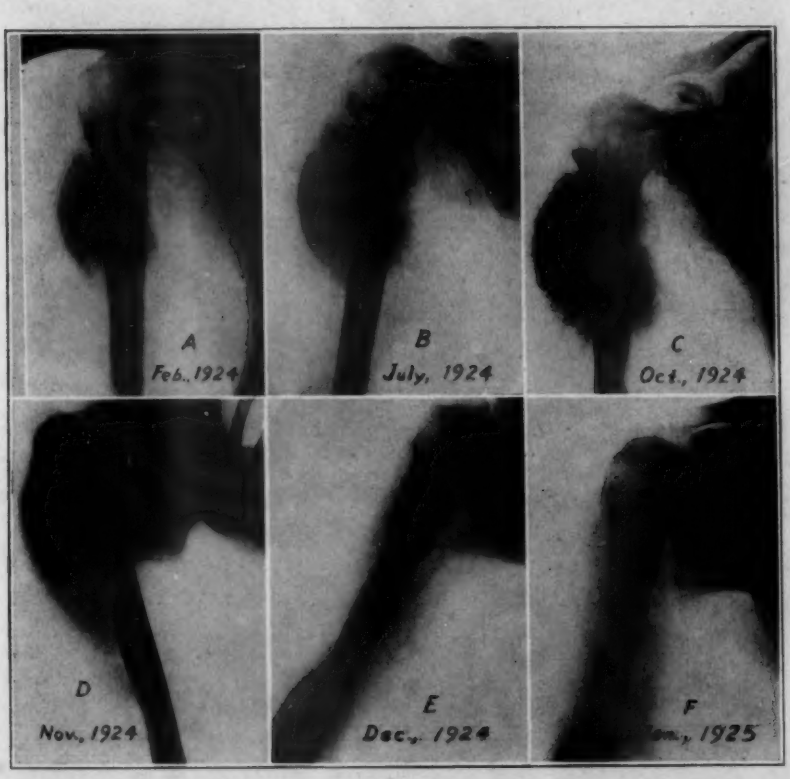

Fig. 2.—*A*, *B* and *C* show the increasing size of the tumor. *D*, *E* and *F* show the appearance following the first operation.

arm. It felt like a pedunculated bony tumor attached to the humerus at one point. The diagnosis at this time was myositis ossificans, and operation was strongly considered, chiefly on account of the increasing pain and also because the mass was steadily increasing in size and growing around the shaft toward the chief blood and nerve supply of the extremity.

The patient was seen by the surgical staff of the Johns Hopkins Hospital, and there was a marked difference of opinion as to the advisability of operating during this actively growing stage. It was decided to await the cessation of growth before operation, unless the pain element or the blockage to the circulation forced surgical steps.

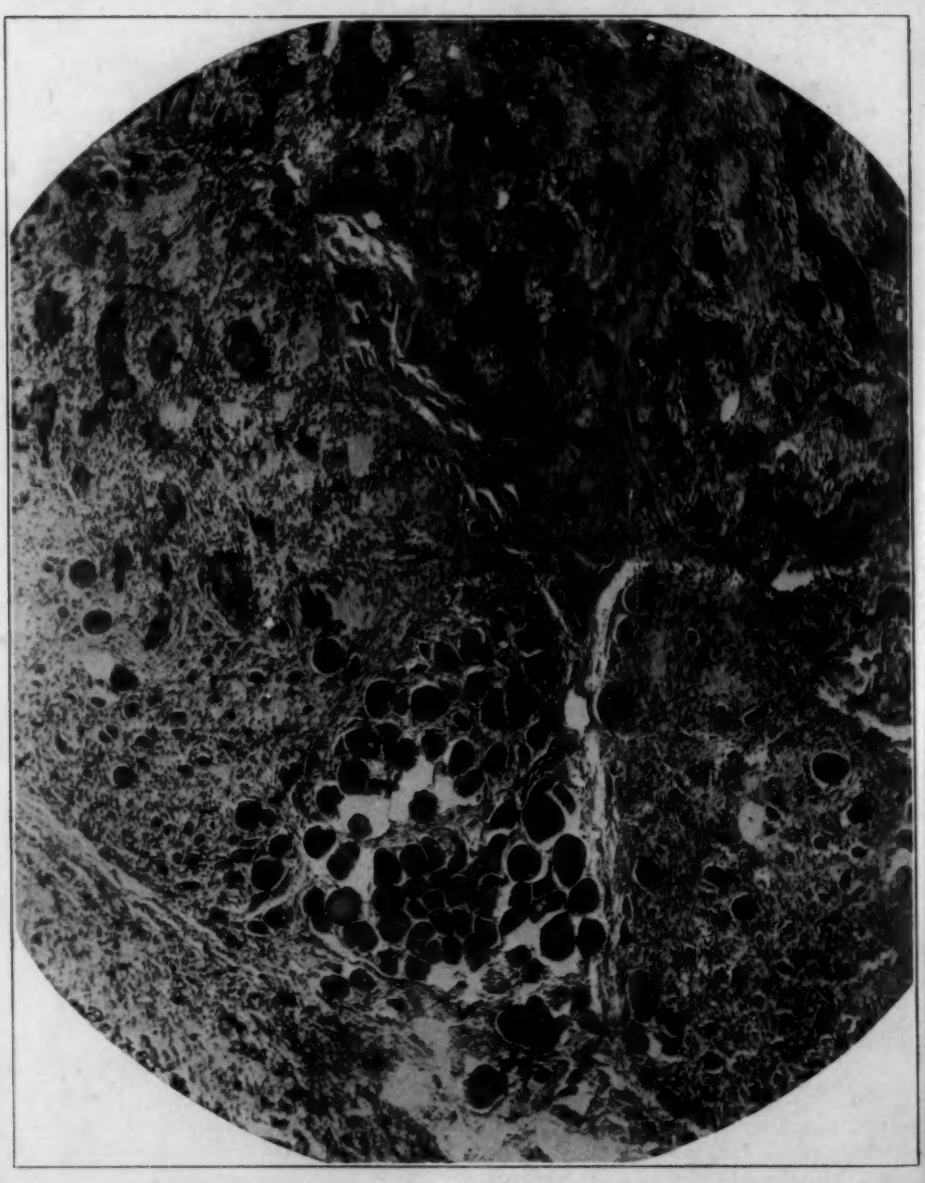

Fig. 3.—Appearance of tissue removed at first operation.

The increase in the size of the tumor was checked by the roentgen ray every five or six weeks (*A, B, C, D, E, F* in fig. 1 and *A, B, C* in fig. 2). The pain was controlled by codeine and the patient allowed to continue his regular work.

Starting about September, 1924, the mass, which had been growing fairly slowly, started a rapid growth and increased as much as 4 cm. in diameter in four weeks, and only a small channel on the inner side of the arm kept the mass from completely surrounding the shaft. Pain was more severe and could not be controlled by codeine. This sudden rapid increase in size made us decide to operate, and operation was done at the Church Home and Infirmary on Oct. 23, 1924, twenty months after injury.

First Operation.—Under ether anesthesia, a large horseshoe-shaped flap of skin was turned up, and a large cartilaginous bonelike mass was exposed; it was reddish-gray and was not encapsulated. There was no line of cleavage, and overlying muscles had to be separated from the growth by sharp dissection. The mass was curetted down to the shaft to which it was intimately connected and which it surrounded, except the groove in which the vessels passed. A good deal of periosteum and some cortex was curetted and chiseled off. The musculospiral nerve was running through the tumor and was knicked badly during the process of separation, but it was sutured at once. The wound was closed, and a firm bandage applied. The wound healed without any complications by December 1. Sections (fig. 3) were made and studied at the Church Home and Infirmary and in Dr. Bloodgood's laboratory at Johns Hopkins Hospital and also by Dr. D. B. Phemister, in Chicago. In none of the numerous sections studied were any signs of malignant disease noted. The sections showed an irregular mixture of bone, cartilage, muscle and connective tissue. In this case the newly formed bone was less orderly than in most cases reported. The trabeculae were blotchy, and the tissue filling the cancellous spaces of the new bone and about its margins were rich in spindle cells.

Following operation, the patient was free from pain, and it was hoped that a cure had been obtained. By January, 1925, two months after operation, some hardness of muscle could be detected clinically and could be definitely seen in roentgenograms taken about this time (*F* in fig. 2 and *A* in fig. 4), and we realized that a recurrence had started. The patient developed complete wrist drop in March, 1925, at which time he was referred to Dr. William Neal (Dr. Kelly's Hospital) for radium therapy. The wrist drop cleared up promptly, but I do not believe this was the result of the radium. The growth increased steadily (*B, C* and *D* in fig. 4) and was extending upward toward the shoulder joint; by September, 1925, the shoulder joint was nearly completely ankylosed, the motions of the elbow, wrist and fingers remained normal, and the patient was practically free from pain. The mass continued to become steadily larger (*E* in fig. 4), and as the blood and nerve supply to the arm were again threatened, amputation was advised. A roentgenogram of the chest did not show growth.

Second Operation.—At the Church Home and Infirmary, July 14, 1926, under ether anesthesia, amputation of the shoulder joint was performed by first sawing through the clavicle, ligating the subclavian vessels in the neck and then injecting 1 per cent procaine hydrochloride into the brachial plexus. The wound healed without complications, and very little postoperative pain was present. Sections showed (fig. 5) typical myositis ossificans and not any evidence of a malignant condition.

I saw and examined the patient in November, 1927, one and one-half years after amputation, and there was no evidence of any further involvement. The patient both looked and said that he "felt fine." He weighed 158 pounds (71.7 Kg.), a gain of 18 pounds (8.2 Kg.) since the date he was first seen.

SUMMARY

This patient was under my care and observation for more than four years. Roentgenograms taken at short intervals make up an important part of the record. The most characteristic roentgenograms are shown (figs. 1, 2 and 4).

The chief factors of interest are:

1. The condition was caused by indirect violence—no blow to the seat of growth but muscular exertion. Probably the stripping of the

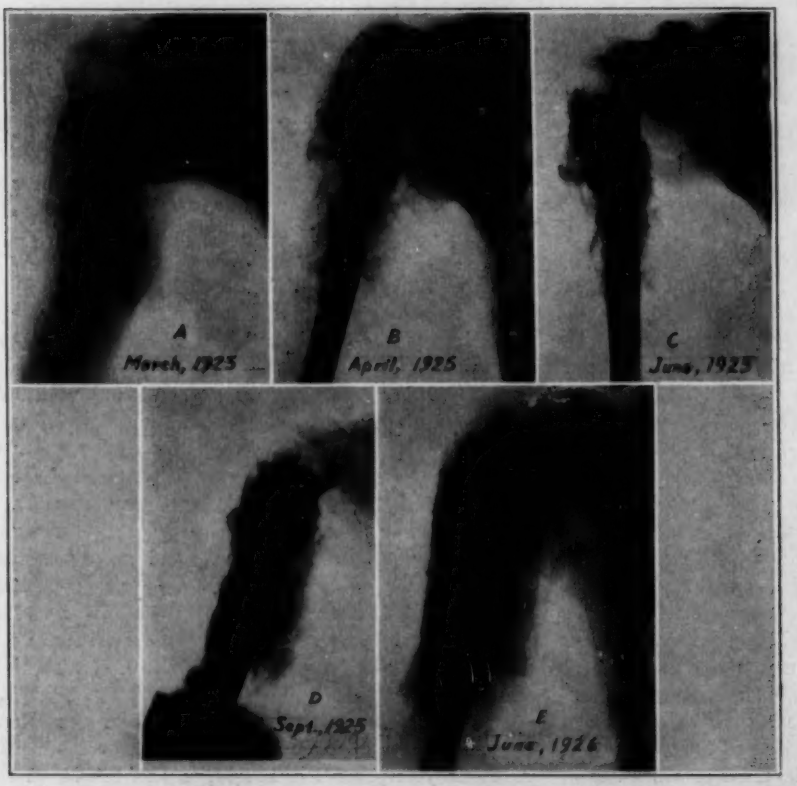

Fig. 4.—*A*, *B*, *C*, *D* and *E* show the recurrence following the first operation.

periosteum and hematoma were the chief etiologic factors, the same factors encountered in a patient with myositis ossificans following backward dislocations of the elbow.

2. A steady painful growth persisted for twenty months, not showing any tendency to become stationary or to recede or a tendency to metastasis. The pain was increasing as the nerves were encroached on.

3. Recurrence occurred following removal. This is the usual history of removal before awaiting the stationary or receding stage.

Fig. 5.—Appearance of tissue removed at amputation.

4. In the differential diagnosis this condition could be confused with periosteal sarcoma, as it resembled it in its steady growth and failure to cease growing; the roentgenograms made prior to the first operation (*A, B, C, D, E, F* in fig. 1 and *A, B, C* in fig. 2) showed the cortex intact and not eroded and irregular as would be expected in sarcoma and, again, the original roentgenograms show better than the

Fig. 6.—Laboratory specimen sawed longitudinally, showing destruction of cortex.

Fig. 7.—Appearance of growth with soft parts dissected away.

reduced prints do, the shadows laid down parallel to the shaft (giving the so-called dotted veil appearance) rather than at right angles as is often noted in sarcoma.

5. The first operation was performed to relieve pain and to excise a steadily growing tumor of twenty months' duration that suddenly became much more rapid in growth; the second operation, amputation

was performed because the shoulder joint became involved and the nerve and blood supply to the extremity were threatened.

6. After the first operation the recurrence, both clinically and from roentgen-ray study (*D, E, F* in fig. 2 and *A, B, C, D E* in fig. 4), more closely resembled sarcoma in its character; the mass was more irregular and was lobulated, which is clearly seen in photographs of dissected specimens (fig. 6); the cortex (figs. 2, 4 and 6) was irregular and eroded (the result of curetting). I believe that a diagnosis of periosteal sarcoma would undoubtedly have been made at this time had one not had the advantages of the early roentgenograms.

MADUROMYCOSIS

FOURTH CASE REPORTED IN THE UNITED STATES *

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Maduromycosis is being encountered in the United States with increasing frequency. The first case was reported by Wright [1] of Boston in 1898; the second by Boyd and Crutchfield [2] of Galveston in 1921, and the third by Gammel, Miskdjian and Thatcher [3] of Cleveland in 1925. The case reported here is the fourth to be reported in the United States.

Previous to 1916, this and other clinically related conditions were known in English-speaking countries as Madura foot or mycetoma. The three cases which have been reported in the United States were described under one of these names. The former name was first used by Colebrook of the Madura dispensary in India, in 1846. The latter term was originated by Carter [4] of London, between 1860 and 1874, who was the first to recognize the fungus nature of this group of diseases. In 1916, Chalmers and Archibald [5] defined mycetoma as a group name embracing the subgroups actinomycoses and the maduromycoses. The actinomycoses are familiar in America, but until recently the recognition of maduromycosis has been rare.

According to the definitions formulated by Chalmers and Archibald, the maduromycoses, although clinically similar to the actinomycoses, differ in respect to the microscopic appearance of the causative fungi. Clinically, both are chronic inflammations manifested by swellings which

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* From the Departments of Surgery and Pathology, University of Minnesota Medical School.

1. Wright, J. H.: A Case of Mycetoma (Madura Foot), J. Exper. Med. **3**:421 1898.

2. Boyd, M. F., and Crutchfield, E. D.: A Contribution to the Study of Mycetoma in North America, Am. J. Trop. Med. **1**:215, 1921.

3. Gammel, J. A.; Miskdjian, H., and Thatcher, H. S.: Madura Foot (Mycetoma); The Black Grain Variety in a Native American, Arch. Dermat. & Syph. **13**:66 (Jan.) 1926.

4. Carter, H. Vandyke: On Mycetoma or the Fungus Disease of India, London, Churchill, 1874, p. 118.

5. Chalmers, A. J., and Archibald, R. G.: A Sudanese Maduromycosis, Ann. Trop. Med. & Parisitol. **10**:169, 1916.

characteristically are perforated by sinuses from which seropurulent, sometimes sanguineous, fluid containing solid or semisolid granules of varying size, shape and color, is liberated intermittently. Pathologically, both conditions are granulomas with minor variations and differences in histologic structure, and on microscopic examination, the granules from each are seen to contain the hyphae of the causative fungi. There are, however, these comparatively recently recognized distinguishing features: whereas the mycelia of the actinomyces are nonsegmented, have poorly defined walls and no chlamydospores, those of the maduromyces are segmented, possess clearly defined walls and usually chlamydospores. Much confusion exists in the recent literature on these diseases, and most of the cases reported were undifferentiated when the reports were made.

REPORT OF A CASE

History.—A negro meat-packer, aged 39, was seen by one of us in the outpatient department of the Minneapolis General Hospital on April 6, 1926. Two days later, he was admitted to the hospital and assigned to the surgical service of the University of Minnesota Medical School. He complained of a swelling of the dorsum of the great toe of the right foot extending to the foot, stiffness of the corresponding metatarsophalangeal joint and discharge from the swelling.

The patient considered the swelling as having resulted indirectly from a contusion of the base of the great toe produced by a falling timber in 1914. At the time of the primary injury, he was employed as a laborer during the construction of a cotton-gin shed on a farm near Trenton, Fannin County, Texas. The injury produced an indentation on the dorsum of the proximal portion of the great toe without causing bleeding or visibly breaking the skin. The part was painful for a time, but after removing the shoe and examining the foot, the patient was able to resume work. Primary swelling and soreness continued for a few days, and the patient soon might have forgotten the injury except for numbness in the toe which persisted for approximately two years. Recurrence of swelling at the site of the original injury was noted in 1916, soon after the return of normal sensation to the part, and approximately ten years before the patient sought admission to the hospital. It consisted of a nodule the size of a "half buckshot," which was firm and movable over the underlying structures, but attached to the skin. The size of the swelling slowly increased during the succeeding seven years, but within the last three years it had doubled in size. The patient attributed the recent comparatively rapid growth to the increased warmth and moisture of the foot induced by the daily wearing of rubber boots at work about a vat of hot water in the meat packing plant. It is interesting to speculate on the correctness of the patient's deduction.

Stiffness of the metatarsophalangeal joint had been present for two years. It was greatest at the time of the intermittent increases in swelling preceding rupture and drainage of a fistula.

Drainage from the swelling had appeared two years previously and had occurred intermittently. The appearance of drainage was usually preceded for two or three days by tenderness over a small area on the surface of the tumor, and it followed spontaneous rupture of the skin at the tender point. The patient described the discharge as "oily, streaked with blood," and as containing black grains which he likened to the cinders which are discharged through the smoke

Fig. 1.—Dorsal view of foot in case of maduromycosis.

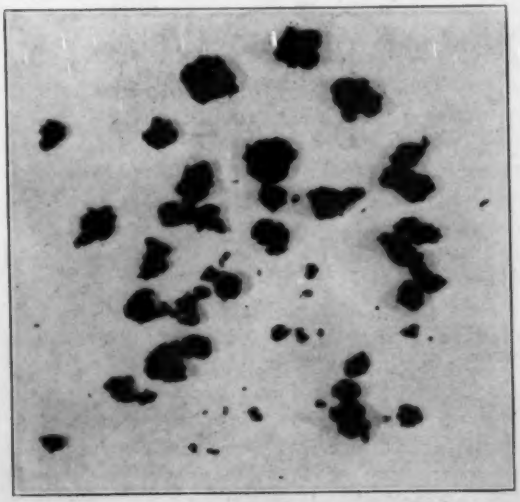

Fig. 2.—Washed brown granules from case of maduromycosis.

stacks of locomotives. By probing with tooth picks, the patient often removed the grains from the fistulas; this procedure was not accompanied by pain. Invariably following drainage for a few days, the fistula closed spontaneously in spite of the patient's efforts to promote drainage by the application of poultices. The greatest amount of drainage which had issued from a single fistula was estimated by the patient at a half teaspoonful.

The patient described the grains as black masses varying from the size of a pinhead to that of a pea, round, rough or angular, soft or firm, heavier than water and resembling cinders.

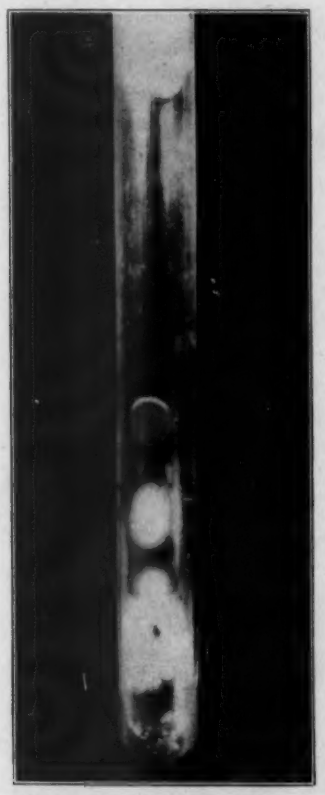

Fig. 3.—Colonies of Madurella ikedai growing on plain agar previously inoculated with washed granules obtained from case of maduromycosis.

The patient was a native of Texas. He had never been outside the United States, but his migrations had included Oklahoma, Missouri, Kansas and Minnesota. He had been a farm laborer for the nine years previous to his work in the packing plant, which was three years before admission to the hospital. (He had not gone barefoot since early childhood.) Otherwise the history was unimportant.

Physical Examination.—Results of general examination were negative, except for swelling of the foot. The temperature and pulse were normal. The swelling was limited to the dorsum and was situated on the distal medial portion of the right foot and the proximal portion of the great toe (fig. 1). It was approximately the size and shape of half a hen's egg. On the surface of the tumor there were

several small nodules. The skin was scarred, smooth and glossy or desquamating, and perforated by several patent or crusted openings from 3 to 5 mm. in diameter. The drainage which issued in small amounts from the patent openings consisted of thin, viscid, turbid fluid streaked with blood and it also contained dark granules having the appearance of coffee grounds (fig. 2). The swelling was firm, except

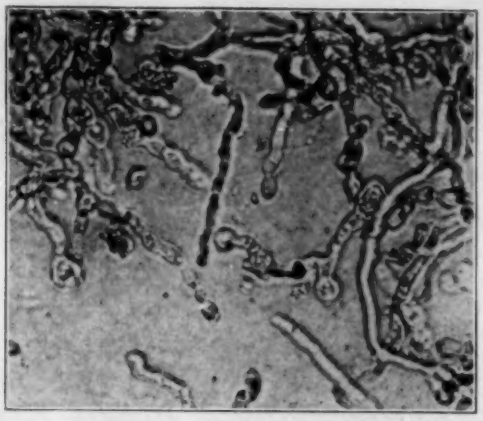

Fig. 4.—Mycelia of Madurella ikedai showing segmentation, distinct walls and chlamydospores.

Fig. 5.—Gross appearance of cross-section of excised tumor from case of maduromycosis showing fibrous and granulation tissue and granules in situ.

for a few small fluctuating areas and there was slight local tenderness. Probing, which did not cause pain, revealed the openings to be the mouths of fistulas. There was neither popliteal nor inguinal adenopathy.

Laboratory Examination.—The urine was normal. The hemoglobin content was 82 per cent; erythrocytes numbered 4,860,000; leukocytes, 6,200, with poly-

morphonuclears, 51 per cent; lymphocytes, 46 per cent; eosinophils, 2 per cent, and basophils, 1 per cent. The Wassermann test on the blood serum was negative. Roentgenograms of the foot were negative. Thick yellow pus expressed from the fistulas contained golden brown granules. Direct smears and cultures of the pus revealed the presence of staphylococci, but tubercle bacilli or fungi were not found. Microscopic examination of the granules, which had been treated with potassium hydroxide, 40 per cent, using the oil immersion lens, revealed a great number of large, segmented mycelia with distinct walls and occasional chlamydospores. On the basis of these data maduromycosis was diagnosed.

Further study of the fungus yielded additional information. Cultures of the washed granules on plain agar were white and of typical powder-puff appearance (fig. 3). On dextrose agar the growths turned brown on standing. The microscopic appearance of wet preparations of the mycelia taken from cultures was identical with those within granules (fig. 4). Suspensions of cultures were injected directly into the lungs, peritoneal cavities and inguinal glands, beneath the skin of the backs and soles and into the veins of several rabbits and guinea-pigs, but the disease was not reproduced in the animals.

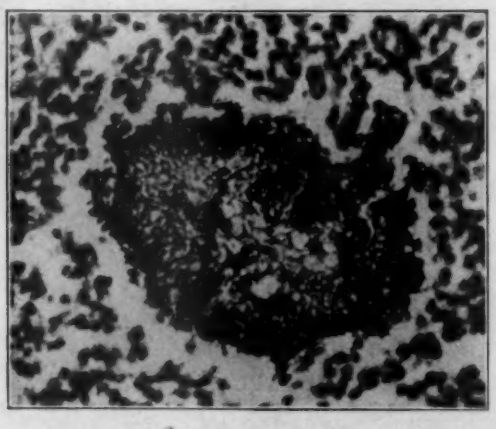

Fig. 6.—Granule occupying abscess cavity.

Treatment and Course.—The treatment was outlined with the collaboration of S. E. Sweitzer of the department of dermatology. The granuloma was excised, and the wound was curetted thoroughly and packed with gauze saturated with 2 per cent copper sulphate solution. Continuous wet copper sulphate dressings were maintained until the wound was well granulated. Several intravenous injections of typhoid vaccine and sterile milk were given as well as a number of local applications of ultraviolet rays. Except following the injections of foreign protein, the patient's temperature remained normal and his general condition excellent throughout the postoperative period. After six weeks, he was dismissed from the hospital with a superficial granulating ulcer at the site of the previous swelling.

Pathologic Examination.—The pathologic data in this case conformed to those of the cases previously reported in the United States, except for a few details. The tumor that was excised from the foot offered gristly, cartillaginous resistance to cutting. On gross inspection, the surfaces thus made were found to be glistening, white, fibrous and mottled, with irregular islands of dark brown granules (fig. 5). The granules were closely packed and were surrounded by a wide inner

zone of soft granulations and a narrow outer zone of yellow tissue. The remainder of the tumor was fibrous tissue. On removal of the granules, abscess cavities with intercommunicating fistulas were exposed.

In microscopic sections, irregular islands of golden brown granules were seen, some loose within abscess cavities (fig. 6), others closely enveloped in granulation and fibrous tissue. The granules corresponded with the descriptions of Wright. They were composed of brown, hyaline-like reticulum and amorphous pigmented material. The periphies of some of the granules were composed of narrow zones of yellow, hyaline, refractile, actively proliferating mycelia (fig. 7). There were

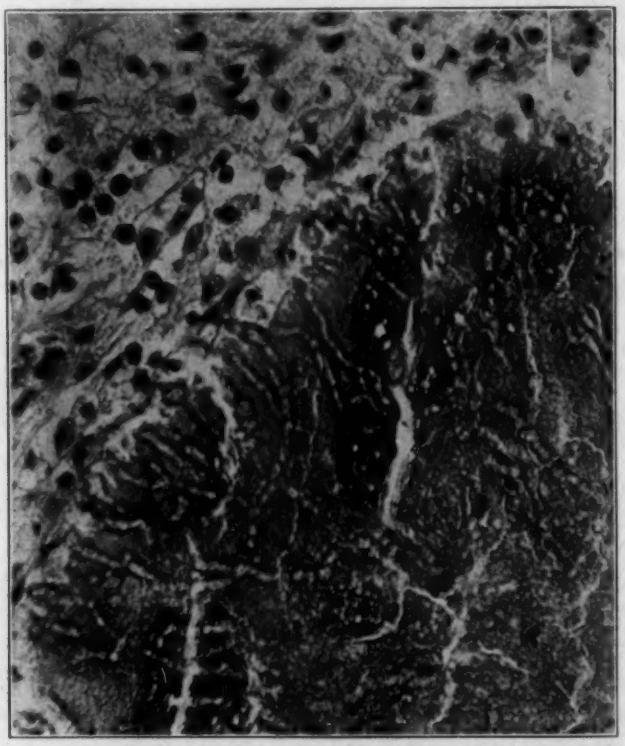

Fig. 7.—Margin of granule; striated zone of proliferating mycelia is shown.

granules the mycelial filaments of which infiltrated the surrounding tissue and others were surrounded by dense fibrous tissue which apparently restricted the growth of the fungus. In the majority of instances, the granules were separated from the surrounding tissue by narrow zones containing few inflammatory cells or broad zones with many cells constituting abscesses.

Mononuclear cells of the plasma cell type predominated and infiltrated the granulation tissue freely. The more remote tissues were markedly edematous and contained not only the cellular infiltrates in large numbers, but also actively proliferating young fibroblasts and new capillaries as apparent by the presence of actively proliferating endothelial cells. Foam cells or xanthoma cells with faintly stained abundant cytoplasm and relatively small nuclei were present in clusters, particularly in the peripheries of abscesses (fig. 8). Giant cells, which are

prominently mentioned by other writers, were absent except for a few irregular, multinucleated foreign-body giant cells near granules in comparatively "dry" fields. In the derma, the sweat glands were atrophied corresponding to the amount of connective tissue in the subcutaneous and underlying tissues. Many of the large and medium-sized arteries, as well as the arterioles, contained thickened intima. Similar changes were noted in some of the small veins.

The fungus which was isolated in this case was extensively studied by J. A. Gammel of Western Reserve University, who was known to be engaged at that time in the detailed study of a fungus which he had isolated in a similar case. A mycologic report of this fungus has already been published with his review of the American cases of maduromycosis.

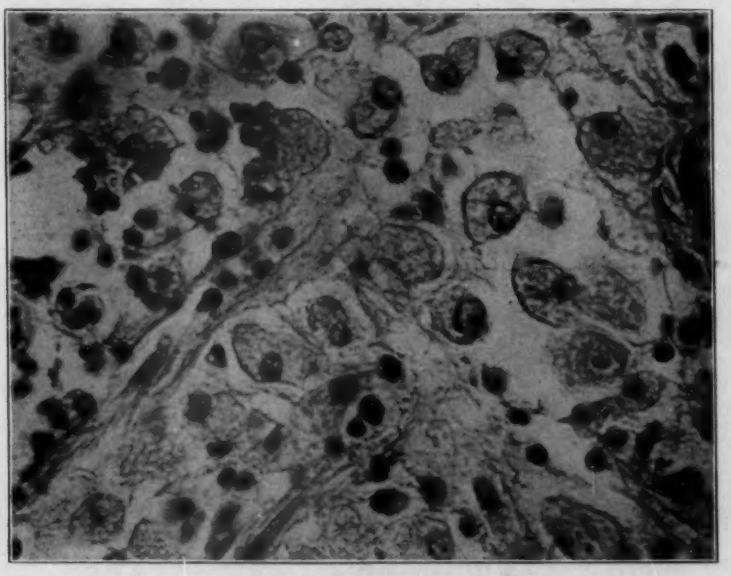

Fig. 8.—Foam cells in granulation tissue.

Gammel concluded that this organism is a new species and defined it as follows:

Madurella ikedai (Gammel 1926[6]): mycelium gray-white, later light brown, darkening certain sugar mediums. Hyphae hyaline or subhyaline, rarely granular, varying in diameter from 1.5 to 5 microns. Chlamydospores numerous only in Sabouraud's glucose broth; in all liquid mediums many hyphae break up into chains of lemoniform or round spores. Acuminate light brown colonies on Sabouraud's and Grütz agar. Sclerotia numerous on surface and in depths of Sabouraud's preservative medium (fig. 9), liquifies gelatin, ferments milk, is thermophile. Pigment production abundant in glucose, gelactose, and maltose, moderate in mannite and levulose, poor in dextrin, poor or absent in inulin, absent

6. Gammel, J. A.: The Etiology of Maduromycosis with a Mycologic Report of Two New Species Observed in the United States, Arch. Dermat. & Syph. **15:** 241 (March) 1927.

in saccharose and lactose broth (fig. 10). Not pathogenic for rabbits or guinea-pigs. Isolated by Ikeda from a maduromycosis with black grains, in Minneapolis.

Gammel made the following statement concerning the identity of this organism:

The complete life history of this organism is not known and as it reproduces by spores which are not contained in asci or basidia it must be placed in Fuckel's class "Fungi imperfecti" or "Hyphomycetes." As no accessory organs of fruc-

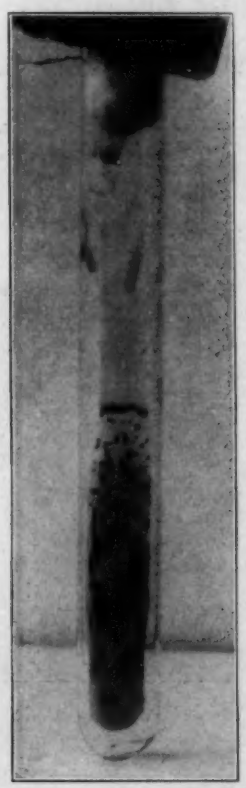

Fig. 9.—Sclerotia numerous on surface and in depths of Sabouraud's preservative medium.

tification can be seen and as reproduction seems to be by means of spores situated on hyphae they belong to Vuillemin's subclass "Hyphales." As the spores are formed by fragmentation of the thallus it must be placed in the order "Thallosporales," suborder "Arthrosporineae," both defined by Vuillemin in 1910. This suborder contains two genera isolated from maduromycoses, namely, genus "Madurella," Brumpt, 1905, and genus "Indiella," Brumpt, 1905.

It is apparent that this organism belongs to the genus "Madurella," Brumpt, 1905, emendavit Pinoy, 1912. On comparison with the characteristics of other species of "Madurella," insofar as available from the literature, the fungus seems to be an undescribed species, at least at the present state of knowledge, and the

name "Madurella ikedai" is proposed for it. As long as the factors which influence permanently the chemical action, biologic behavior and the macroscopic and microscopic aspects of these molds are not known the establishment of new species is justifiable when organisms are found which differ in essential points from known species.

Subsequent Course.—On recent examination, Sept. 5, 1927, the patient was found to be in excellent general health. He works at his trade daily. The surgical wound has been healed for fifteen months. At the site of the original swelling

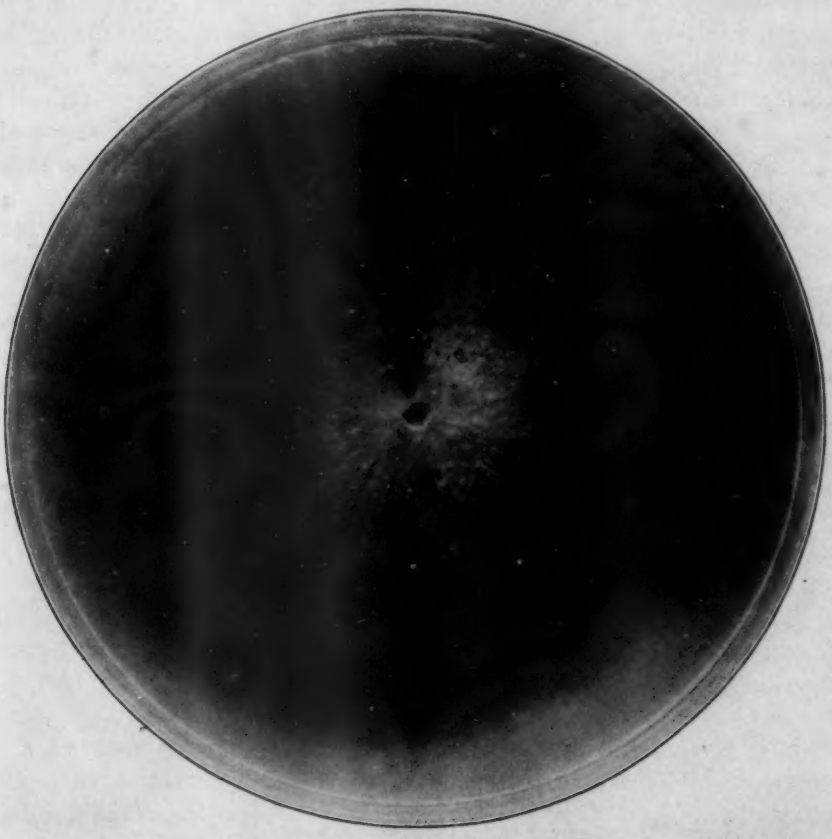

Fig. 10.—Giant colony of Madurella ikedai (Gammel 1926) on 1 per cent dextrose agar, 2 weeks old. The fungus grows now, one and a half years after isolation, much faster than in the first generations. The dark granules near the center are sclerotia. The agar is deep brown except in the extreme periphery of the plate (natural size).

recurrent nodules and fistulas are present. Granules were recovered from the drainage material. Slight tenderness on the plantar surface of the foot indicates that extension of the inflammatory process in that direction has occurred. The patient does not feel any discomfort while the sinuses are open and the drainage is free. It is probable that amputation ultimately will be required to eradicate the disease process.

THE PRESENT STATUS OF MYCETOMA

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The term mycetoma is used at the present time to include maduromycosis and actinomycosis. In 1846, before the causes of these chronic inflammatory conditions were known, Colebrook [1] first recorded in the literature the geographico-anatomic name, Madura foot. Carter,[2] in his monograph published in 1874, designated them on the basis of etiology and pathology by the term mycetoma (meaning fungus tumor). The term maduromycosis was introduced in 1916 by Chalmers and Archibald [3] to apply to an etiologic subgroup of mycetoma, which they distinguish from actinomycosis. Actinomycosis, as caused by *Actinomyces bovis,* has been known since the work of Bollinger in 1878, and as many of the organisms isolated from cases of Madura foot or mycetoma of the foot have been found to belong to the genus *Actinomyces,* actinomycosis likewise has become a subgroup of the mycetomas. Although progress has been made during the last decade in respect to the etiologic and mycologic classification of mycetoma, much confusion still exists in the literature, from botanic considerations involved, which has tended to obscure the clinical and pathologic aspects of the mycetomas, as well as the fact that as yet no differentiation on a clinical or pathologic basis can be made of the varieties of mycetoma. It is necessary, therefore, in any such consideration of these diseases, to retain the term mycetoma as the name of the group.

Twenty-one cases of mycetoma have been reported from the United States. After a critical study of these cases, Gammel [4] concluded that according to Chalmers and Archibald's criteria, most of them were cases of actinomycosis and that only three were cases of maduromycosis.

It is my purpose in this paper, in contrast to that of most recent writers, to present a consideration of the mycetomas from a clinical rather than from an etiologic standpoint. It was my privilege to encounter the fourth case of maduromycosis in the United States, which was the first case to be reported as such.[5]

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1. Chalmers, A. J., and Christopherson, J. B.: A Sudanese Actinomycosis, Ann. Trop. Med. & Parasitol. **10**:223, 1916.

2. Carter, H. Vandyke: On Mycetoma or the Fungus Disease of India, London, Churchill, 1874, p. 118.

3. Chalmers, A. J., and Archibald, R. G.: A Sudanese Maduromycosis, Ann. Trop. Med. & Parasitol. **10**:169, 1916.

4. Gammel, J. A.: The Etiology of Maduromycosis with a Mycologic Report of Two New Species Observed in the United States, Arch. Dermat. & Syph. **15**:241 (March) 1927.

5. Thompson, H. L., and Ikeda, Kano: Maduromycosis: Fourth Case Reported in the United States, Arch. Surg. (In press.)

SYNONYMS AND NOMENCLATURE

The conditions referred to in this paper as the mycetomas have been discussed under many names. According to Castellani and Chalmers,[6] the natives of India designated them by the vernacular terms, "keerenagrah" and "kirudeo," which appear to have been suggested by the likeness of the tumors and fistulas to the dwellings of worms, and also by the expression "ghootloo mahdee," probably because of the resemblance of the grains to the eggs of insects. The mycetomas were confused with tuberculosis by some of the early writers; cases were described as "morbus tuberculosis pedis," by Godfrey[8] in 1844, and as "Godfrey and Eyre's tubercular disease," by later writers. In 1846, Colebrook[7] recorded the name Madura foot as that commonly applied to mycetoma of the foot in and near Madura, a town in the southern part of Deccan, India. In 1874, H. Vandyke Carter gave these diseases the scientific name "mycetoma" (Gr. μύκης fungus, ὦμα tumor).[5] In 1913, Pinoy[8] first used the name mycetoma inclusively for the etiologically distinct conditions, the true mycetomas and the actinomycoses. In 1916, Chalmers and Archibald also used mycetoma inclusively for the subgroups actinomycosis and maduromycosis. The term maduromycosis thus replaced the name "true mycetoma" of Pinoy. This nomenclature has been adopted by Castellani and Chalmers, Brumpt,[9] Langeron[10] and Gammel.

DEFINITION OF TERMS NOW IN USE

Previous to the acceptance of Chalmers and Archibald's classification of the mycetomas, the term mycetoma had a restricted meaning in that it was the name applied to all the granulomas of fungous origin that occurred in the foot without consideration of the type of causative fungus. With the proposal, in the light of added knowledge, of an etiologic classification of this group of similar diseases, these investigators retained the term mycetoma as a group name and defined it as follows:

Mycetoma includes all growths and granulations in any of the tissues of man or animals, which are caused by fungi belonging to different genera and species, which produce bodies, called grains, composed of hyphae and sometimes chlamydospores and which are found either imbedded in the pathological tissues or escaping in the discharge therefrom.

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6. Castellani, A., and Chalmers, A. J.: Manual of Tropical Medicine, ed. 3, New York, William Wood & Co., 1919, pp. 2110-2149.

7. Colebrook, cited in footnotes 1 and 3.

8. Pinoy, E.: Actinomycosis et mycétomes, Bull. de l'Inst. Pasteur **11**:929 and 977, 1913.

9. Brumpt, Emile: Précis de parasitologie, ed. 3, Paris, Masson, 1922, pp. 965-969.

10. Langeron, Maurice: Mycétomes, in Nouveau traité de médicine, Paris, Masson, **4**:445, 1925.

This is the accepted use of the term at the present time.

The mycetomas were divided by Chalmers and Archibald into two groups: (*a*) the maduromycoses, and (*b*) the actinomycoses. These terms also were defined by them as follows:

> The maduromycoses are the forms of mycetoma with grains composed of large segmented mycelial filaments possessing well-defined walls and usually chlamydospores. . . . The actinomycoses are those forms of mycetoma with grains composed of very fine nonsegmented mycelial filaments, the walls of which are not clearly defined from the contents and in which chlamydospores are absent.

In order to avoid further confusion of the term grain with the nonspecific term granule, and with the mycologic expression sclerotium, the former term also was defined:

> The word grain is applied to certain bodies found in mycetomas of varying color, consistence, size and shape, which are composed of hyphae, and sometimes chlamydospores, imbedded in a matrix and which on germination give rise to mycelial filaments.

The term grain thus has become a specific term for the bodies found in the tissues and discharges of the mycetomas as the word tubercle is for the pathologic unit of tuberculosis.

Chalmers and Archibald contributed much to the elucidation of the subject of mycetoma, although considerable confusion continues to exist.

HISTORY

In an attempt to clarify the confusion in the literature on the mycetomas of up to a decade ago, Chalmers and Christopherson employed the helpful method of dividing their discussion of the evolution of the knowledge of this group of diseases into four historic periods. When possible, they designated each period by the most significant name for the diseases extant during that period. With the same object in mind, I wish to modify the name of their fourth period and add a fifth period to their list. This modification of their plan is as follows: (1) The early period (—1846); (2) the Madura foot period (1846-1860); (3) the mycetoma period (1860-1878); (4) the mycetoma-actinomycosis period (1878-1913), and (5) the mycetomas period (1913-). My fifth period dates from the extension of the term mycetoma from its original conception as one disease to its present status as a group name for several similar diseases.

The early period is characterized by an attempt to establish the first record of cases in this group of pathologic conditions. According to Chalmers and Christopherson, the various types of mycetoma of the foot were distinguished from elephantiasis and possibly from yaws by the early Indian surgeons, provided Waring's reference to Sanscrit

writings is correct. Kaempfer is credited by many with being the first to describe them in 1712, using the name perical, but Chalmers and Christopherson have pointed out that this term was applied to any enlargement of the foot, including yaws and elephantiasis. In 1714, a missionary of Pondichery described a disease of the foot which may have been mycetoma. There is some evidence that Heyne [1] saw a mycetoma in 1806, and Brett's [1] adipose sarcoma, described in 1840, likewise may have been a mycetoma. Although the disease probably was known before the close of this period, it is difficult to establish the first record of it in the literature.

The Madura foot period, between 1846 and 1860, represents the time of definite establishment of mycetoma of the foot as a clinical entity and the period during which some effort was made to establish its identity etiologically. The association in the literature of the disease, mycetoma of the foot, with the town, Madura, was first made by Gill [1] of the Madura dispensary, in 1842, when he called attention to a peculiar disease of the foot. The name Madura foot, however, was first recorded by Colebrook,[1] Gill's successor, in 1846; he stated that this was the current name for it in certain parts of India. Between 1845 and 1855 grains were described or illustrated by von Langenbeck [1] from a case of spinal caries, by Lebert [1] from pus from the thoracic wall, by Ballingal [1] from a tumor of the jaw, and by Paget and Smith [1] from a swelling of the foot, all of which probably were actinomycosis. In 1856, Godfrey,[3] at Bellary, described a case of what he believed to be tuberculosis of the foot with accidental black deposits, and he named it morbus tuberculosis pedis. In 1858, Rustomji [3] recognized varieties of Madura foot represented by black and yellow grains. In 1860, Eyre [3] gave the first complete review of the existing knowledge of Madura foot in which he referred to the presence in the tissues of "tubercles." During this period, mycetoma of the foot became known as Madura foot and by many was thought to be tuberculosis.

The mycetoma period began in 1860 when Carter [1] began his series of publications on Madura foot. Taking note of the various colors of grains, he originated on this basis the first classification of Madura foot. He distinguished black or melanoid, yellow or ochroid, and red varieties of the disease. He also recognized the fungous origin of Madura foot, but erred in considering the yellow form a degenerative stage of the black, and in maintaining that an organism called *Chinonyphe carteri* was the cause of the disease. Carter renamed the disease which he had described, and in 1874, published a monograph, "On mycetoma or the fungus disease of India." Lewis and Cunningham,[3] in 1875, and Berkeley,[3] in 1876, showed that the chinonyphe described by Carter was not the cause of mycetoma. In 1886, Carter [3] withdrew his contention regarding the causative organism and likened

the fungus of mycetoma to that of actinomycosis. This period somewhat overlaps the succeeding period wherein the nature of certain types of actinomycosis became established.

During the actinomycosis-mycetoma period, from 1878 to 1913, little real progress was reported, and the knowledge that was contributed was obscured by lack of uniformity in nomenclature. The period dates from the work of Bollinger,[1] the results of which were published in 1876, on lumpy jaw of cattle, in which a branching fungus was constantly observed. The organism was studied by Hartz [1] who gave it the name *Actinomyces bovis* (1877). As pointed out by Gammel, the British object to the use of this name and apply the generic name *Nocardia* after the French pathologist Nocard, on the authority of the First International Congress of Pathology. The French, on the other hand, call the genus *Discomyces,* but the Germans and Americans continue to call it *Actinomyces,* the latter accepting the authority of the Society of American Bacteriologists. Many other generic names have been suggested by investigators of organisms from isolated cases. Many of the organisms with different names have been found to be identical and accordingly have been reclassified under one or the other group.

It was during this period that Carter [8] abandoned his contention that *Chinonyphe carteri* was the cause of mycetoma. In 1893, Kanthack [11] studied both yellow and black mycetomas, concluded that the yellow variety was actinomycosis, and, in direct opposition to Carter's earlier view, held that the black was a degenerative stage of the yellow. Unna [8] examined Kanthack's specimens and found many distinctions between the fungi of the two varieties. Finally, in 1894, Boyce and Surveyor [12] established definitely the existence of different organisms in black and yellow types of mycetoma of the foot.

In 1894, Vincent,[1] in Algiers, isolated from a mycetoma with yellow grains an organism which he called *Streptothrix madurae.* Notwithstanding the fact that this generic name had been used previously for an entirely different organism, the term continues to appear in the literature as a synonym for *Hyphomycetes* or *Actinomyces,* only to further the confusion already existing.

Toward the close of this period distinct progress was made by a few workers in the identification and classification of the fungi isolated from the granules taken from cases of this peculiar group of diseases.

The mycetomas period dates from 1913, when Pinoy proposed to extend the term mycetoma to include actinomycoses, caused by *Actino-*

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11. Kanthack, A. A.: Madura Disease (Mycetoma) and Actinomycosis, J. Path. & Bacteriol. **1**:140, 1893.

12. Boyce, R., and Surveyor, N. F.: Upon the Existence of More Than One Fungus in Madura Foot (Mycetoma), Phil. Tr. Roy. Soc., London **185**:1, 1895.

myces, and true mycetoma, caused by true molds. After intensive research and exhaustive review of the literature, Chalmers and Archibald, in 1916, clearly defined the mycetomas and the subgroups and differentiated similar conditions. In a recent report, Gammel listed thirteen species of *Actinomyces* isolated from cases of mycetoma of the foot, and nineteen species of fungi isolated from maduromycoses, including one which he isolated and another which he studied, isolated from the case which Ikeda and I reported.[5]

A number of cases of mycetoma have been observed in the United States. The first case of mycetoma was reported in 1896 by Hyde, Senn and Bishop.[13] In all, twenty-two cases have been recorded in the literature. According to Gammel, who made a critical study of the cases, only four are of maduromycosis. The first case of maduromycosis was reported by Wright [14] of Boston in 1898, the second by Boyd and Crutchfield [15] of Galveston in 1921, the third by Gammel, Miskdjian and Thatcher [16] of Cleveland in 1925, and the fourth by me in collaboration with Ikeda [5] in 1927. Boyd and Crutchfield's case was of the white grain type, and the others of the black grain type. The fact that two thirds of the cases observed in the United States have been reported within the last decade from many sections of the country indicates that this group of diseases is beginning to arouse widespread interest.

Although the subject of the mycetomas is still in a confused state, it is obvious that the efforts of Carter, Pinoy, and Chalmers and Archibald have simplified it considerably. Carter recognized the fungous nature of the mycetomas, and the possibility of their being tuberculous in origin was thereby excluded. Pinoy laid the foundation for the work of Chalmers and Archibald whereby the mycologic distinction between the actinomycoses and maduromycoses was set forth clearly. The actual confusion which continues to exist in regard to the etiology of the mycetomas is chiefly botanic and persists largely because the entire subject of mycology is more or less in a confused state. For a clearer understanding of the etiology of this interesting group of diseases further elucidation of the life histories of the causative organisms is necessary.

13. Hyde, J. N.; Senn, Nicholas, and Bishop, D. D.: A Contribution to the Study of Mycetoma of the Foot as It Occurs in America, J. Cutan. Dis. **14**:1, 1896.

14. Wright, J. H.: A Case of Mycetoma (Madura Foot), J. Exper. Med. **3**:421, 1898.

15. Boyd, M. F., and Crutchfield, E. D.: A Contribution to the Study of Mycetoma in North America, Am. J. Trop. Med. **1**:215, 1921.

16. Gammel, J. A.; Miskdjian, H., and Thatcher, H. S.: Madura Foot (Mycetoma); the Black Grain Variety in a Native American, Arch. Dermat. & Syph. **13**:66 (Jan.) 1926.

ETIOLOGY

The mycetomas are widely distributed throughout tropical and subtropical zones. They are endemic in certain districts in India and are seen occasionally in Africa, Europe, South America, Mexico and the United States. In this country twenty-two cases [4] have been reported previously, including one each from Indiana, Iowa, Massachusetts, Maryland, and Minnesota, two from Arizona, three from California, and the remainder from Texas.

On the basis of an analysis of 100 cases of mycetoma, the majority of which were said to be maduromycoses, Bocarro [17] of Hyderabad, in 1893, pointed out the chief factors which predisposed to these fungous infections. Ninety-two of the patients were males and eight females. The patients were from 12 to 80 years of age, most of them being between 21 and 40. All of the patients were rural inhabitants, and ninety-one tilled the soil. Seven of the eight female patients were wives of agriculturists. The foot was the site of the disease in ninety-three cases, the leg in two, the hand in three, and the trunk in two. Carter emphasized the fact that none of his patients with mycetoma had ever worn shoes and that most of them attributed the origin of the disease to trauma of the barefoot and many specifically to thorn pricks. Boyd and Crutchfield, in an analysis of the cases which occurred in North America, corroborate the observations of Bocarro and Carter.

The mode of inoculation is not known. In view of the fact that a history of trauma is obtainable in about one half of the cases, it is presumable that the fungus enters through an open wound. The hair follicles and the ducts of the glands of the skin also have been suggested as points of entrance. The mycetomas are most common, therefore, in tropical and subtropical zones, among men between the ages of 20 and 40 who till the soil and who have received an injury to the bare foot.

A characteristic of the mycetomas is the presence in the tissues and discharges of granules or grains which contain the causative fungus. The several colors of grains were noted by Carter, who employed this feature as the basis for the earliest classification of the mycetomas. He distinguished the yellow or ochroid, the black or melanoid and the red varieties. Within the last two decades, added knowledge of the fungi which produce the mycetomas has permitted the formulation of the other classifications on a mycologic basis. Thus under the group name mycetoma Pinoy included the actinomycoses and the true mycetoma, Brumpt included the actinomycotic mycetoma and the true mycetoma, and Chalmers and Archibald included the actinomycoses and the maduromycoses.

17. Bocarro, J. E.: An Analysis of One Hundred Cases of Mycetoma, Lancet **2**:797, 1893.

The grains of the mycetomas vary in respect to color, consistency, size and shape. Those of the pale or ochroid variety are white, gray or yellow; those of the melanoid, brown or black. There is also a rare variety in which the grains are red. The grains in some cases are soft and of cheesy consistence, in others firm, hard or brittle. The latter may be softened in caustic potash or eau de Javelle. Ordinarily they are about 1 mm. in diameter, but they commonly exist in aggregations which are several millimeters in diameter. Their shapes vary as irregularly spherical, cuboidal, lobate or moruloid, and their surfaces are smooth, faceted or bossy. The yellow grains frequently are said to resemble fish-roe, and the black, gunpowder, coffee grounds, pieces of coal or cinders.

Microscopic examination reveals the structure of the grains. The elements contained are mycelia, pigment granules and leukocytes. Often three zones are demonstrable: a central zone, composed of a mycelial reticulum and a varying number of pigment granules, an intermediate zone, irregular, amorphous, and deeply pigmented, and an outer zone, refractile, striated and composed of hyphae, usually disposed radially. Leukocytes are present in all zones. The staining qualities of the grains vary.

In teased specimens of soft or softened grains the individual hyphae may be studied, and their characteristics distinguished. The mycelia in the maduromycoses are large segmented filaments, with walls which are well defined, and usually chlamydospores of varying sizes and shapes. The mycelial filaments of the actinomyces do not have clearly defined walls and are nonsegmented and chlamydospores are absent. The microscopic appearance is the basis for differentiation of the actinomycoses and the maduromycoses.

Growth cultures made from the grains on ordinary mediums and incubated at 37 C., usually appear in a few days in the form of tufts of delicate white filaments. In cultures made from the black grains on dextrose mediums, a rich brown pigment gradually spreads throughout the medium. The nature of the pigment in the grains or in the medium is not known. As pointed out by Gammel, it has been known for some time that each color of grain may be produced by widely different fungi, and it was recently shown that the same species may produce different colors of grains. Therefore the classification of the mycetomas on the basis of the color of the grains is of clinical interest, but of no diagnostic value. Reproduction of the mycetomas in animals has been successful in a few instances.

An excellent recent study of the etiology of the mycetomas in the United States was reported by Gammel. He enumerates thirteen species of the genus *Actinomyces* which have been isolated in cases of actinomycosis, and nineteen species, belonging to two classes and eight genera,

isolated in cases of maduromycosis each of which produced the clinical picture of mycetoma of the foot. The following list of organisms was taken from this report:

Organisms isolated in cases of actinomycosis:

I. Class Schizomycetes
- A. Genus: Actinomyces
  1. *Actinomyces bovis,* Harz, 1877.
  2. *Actinomyces mexicanus,* Boyd and Crutchfield, 1921.
  3. *Actinomyces asteroides,* Eppinger, 1890.
  4. *Actinomyces bahiensis,* Pirajá da Silva, 1919.
  5. *Actinomyces convolutus,* Chalmers and Christopherson, 1916.
  6. *Actinomyces indicus,* Kanthack, 1893.
  7. *Actinomyces madurae,* H. Vincent, 1894.
  8. *Actinomyces somaliensis,* Brumpt, 1906.
  9. *Actinomyces sp.,* Yazbek, 1920. (Seems closely related to *Actinomyces somaliensis.*)
  10. *Actinomyces sp.,* Yazbek, 1920. (Related to *Actinomyces asteroides.*)
  11. *Actinomyces pelletieri,* Laveran, 1906.
  12. *Actinomyces verrucosus,* Miescher, 1916.
  13. *Actinomyces ponceti,* Verdun, 1912.

Organisms isolated in cases of maduromycosis:

I. Class: *Fungi imperfecti*
- A. Genus: Madurella
  1. *Madurella mycetomi,* Laveran, 1902.
  2. *Madurella bovoi,* Brumpt, 1910.
  3. *Madurella oswaldoi,* Parreiras Horta, 1919.
  4. *Madurella ramiroi,* Pirajá da Silva, 1919.
  5. *Madurella tabarkae,* Blanc and Brun, 1919.
  6. *Madurella tozeuri,* Nicolle and Pinoy, 1908.
  7. *Madurella americana,* Gammel, 1925.
  8. *Madurella ikedai,* Gammel, 1926.
- B. Genus: Indiella
  1. *Indiella mansoni,* Brumpt, 1906.
  2. *Indiella reynieri,* Brumpt, 1906.
  3. *Indiella brumpti,* Pirajá da Silva, 1922.
- C. Genus: Glenospora
  1. *Glenospora khartoumensis,* Chalmers and Archibald, 1916.
  2. *Glenospora semoni,* Chalmers and Archibald, 1917.
- D. Genus: Scedosporium
  1. *Scedosporium apiospermum,* Saccardo, 1911.
  2. *Scedosporium scleriotiale* (seu nigricans), Pepere, 1914.

II. Class: Ascomycetes
- A. Genus: Allescheria
  1. *Allescheria boydii,* Shear, 1921.
- B. Genus: Aspergillus
  1. *Aspergillus buoffardi,* Brumpt, 1906.
- C. Genus: Sterigmatocystis
  1. *Sterigmatocystis (Diplostephanus) nidulans,* Eidam, 1883, variety Nicollei, 1906.
- D. Genus: Penicillium
  1. *Penicillium mycetogenum,* Mantelli and Nigri, 1915.

Thus a total of thirty-two species have been isolated in cases of mycetoma reported from the United States. It is remarkable that a uniform clinical and pathologic picture is produced by so great a variety of fungi.

Gammel pointed out that knowledge of the mycology of most of these organisms is limited and the nomenclature and classification therefore must remain provisional and rather artificial. Fungi parasitic to man are difficult to identify in many cases because they lack the morphologic characters by which the identities of other fungi are determined. These molds are identified, therefore, not only by cultural characteristics but also by pathologic behavior. Many fungi are subject to great variations, and the distinction between new species and permanent variations can be made only after prolonged study. The manner in which some fungous parasites occur in nature is not known. Many of the genera are not well defined and the descriptions of many of the species are incomplete. It is as yet not known in many instances whether differences in color, pigment formation, and the formation of grains are constant features or individual variations resulting from external conditions. Ability of a given species to ferment certain sugars may be either lost or acquired. In respect to the fungi imperfecti, at least, the life histories are imperfectly known, but when in instances they are established they are removed from this class and reclassified with the appropriate groups. For recent classifications of all known species of actinomyces reference may be made to Bergey's [18] manual and to that of Chalmers and Christopherson.[1]

PATHOLOGY

Regardless of the existence of this multiplicity of causative organisms, the gross and microscopic pathology of the several varieties of mycetoma is the same except in minor details. The essential pathologic features of these fungous tumors are those which are characteristic of the infectious granulomas, including slowly progressing local necrosis and abscess formation, intermittent discharge through fistulous tracts of peculiar fluid containing characteristic grains, and replacement of the normal structures by granulation and fibrous tissue. In view of the uniformity of pathologic characteristics of the mycetomas, any clinical or pathologic consideration of one or more of them must apply to the entire group.

In man the growth of the fungi is usually sluggish; this permits extensive local reaction. In the tissues immediately surrounding the fungus, liquefactive degeneration takes place, and in this fluid the grains appear to be formed by an aggregation of fungus elements. The tissue reaction throughout the course of the disease remains local and is

18. Bergey, D. H.: Bergey's Manual of Determinative Bacteriology, ed. 2, Baltimore, Williams & Wilkins Company, 1925, pp. 345-398.

restricted to round cell infiltration, in many cases the formation of giant cells, in some of the appearance of eosinophils, and in all instances the production of granulation tissue and extensive scarring. The liquid and granules are ultimately discharged through fistulas.

The gross manifestations are local or general tumefaction of the affected part and corresponding degrees of deformity. The skin is of normal or darker hue and in cases of long standing mottled with scars. The surfaces of the swellings are studded with nodular or fungating elevations and perforated by irregular pouting openings. From these openings there is discharged spontaneously, or there may be expressed, a peculiar viscid seropurulent fluid with, as the case may be, black, yellow or red grains. A probe introduced into them follows a tortuous course into the depths of the part. Secondary infection alters the appearance correspondingly.

The affected tissues, including bone, slightly resist cutting. The surfaces of sections made by cutting through the tumors are yellow-gray and moistened by the peculiar fluid. The anatomic structures are slightly changed or indistinguishable, depending on the duration of the disease. Muscle and bone, and, less commonly, fascia, tendons and joints are involved to a greater or lesser degree. Any of these tissues may be the site of cellular infiltration, or they may be replaced by gray or yellow oleaginous or myxomatous masses by embedded aggregations of pigmented fungous masses or by abscess cavities. The fistulas usually contain grains; they intercommunicate or extend through tortuous courses to the surfaces of the swellings.

The microscopic features are those of the infectious granulomas, with minor individual variations. The tumors are composed of granulation and fibrous tissue with abscess cavities and fistulas interspersed. At intervals endarteritis, periarteritis and small hemorrhagic areas are commonly found. Compact aggregations of grains are occasionally found in the tissues, but more commonly in the abscess cavities and fistulous tracts. Old fistulas which have discharged their content are filled with round cells; unruptured fistulas contain, besides the grains, fluid in which are suspended polymorphonuclear leukocytes and cellular detritus. The walls of the fistulas and abscesses are composed of central linings of granulation tissue containing young connective tissue fibrils, and older connective tissue peripherally. Centrally there are round cells about the cavities and fistulas, and remotely there are usually epithelioid and giant cells.

A typical microscopic section made by cutting through an abscess cavity or a fistulous tract appears as follows: The grain composed of mycelial elements, pigment and débris is suspended in fluid which fills the cavity of the abscess or fistula. Suspended in the fluid also are polymorphonuclear leukocytes and cellular débris. A zone of tissue

composed of connective tissue fibrils, vascular loops and small round cells constitutes the lining of the cavity or tract. External to this is a more or less distinct intermediate zone of large round and giant cells, and a peripheral zone of dense connective tissue. In some of the cases reported, eosinophils have been present. The grains are described elsewhere in this article.

SYMPTOMS

The clinical aspects and symptoms, as well as the pathologic characteristics of the several mycetomas appear to be identical from the standpoint of present knowledge. Thus from a clinical standpoint, what is said of one, applies to the group.

As the means by which the pathogenic fungi of the mycetomas enter the tissues is not known, statements in regard to the incubation period are merely speculative. Usually it is said to vary greatly or to range from several days or weeks to as long as three months. Such statements are based on the assumption that the fungi gain entrance to the tissues through open wounds. A history of previous trauma to the affected part can be secured in probably not more than half the cases, and, when obtainable, antedates the onset of symptoms for a period sufficient to permit healing of the wound. When a history of trauma is obtained it usually is estimated to have occurred from several weeks to as long as several years before the onset of symptoms.

Prodromal symptoms may be absent, or presaging actual tumefaction for a period of a few days or weeks, there may be local tenderness or slight redness or discoloration of the skin.

The modes of onset of the diseases are neither uniform nor characteristic. Carter enumerated four modes of first appearance of the mycetomas as: (1) a papule which may be livid or mottled, but which is neither painful nor feverish; (2) a nodule which is deep-seated and fixed; (3) a vesicle surmounting a swollen, hard area, or (4) an abscess which on rupture is found to be a fistula. The most common mode of onset is in the form of a nodule.

In well established cases, swelling is the most prominent symptom. It usually appears first as a small round nodule from 0.5 to 1 cm. in diameter on either the dorsum or the sole of the foot. Subsequently other nodules make their appearance near the first, where the intervening tissues then appear more or less swollen. In advanced cases the entire foot may be tumefied and its surface studded with nodules or fungous excrescences.

Variable degrees of deformity result from the swelling. It varies from local tumefaction to general increase in the size of the foot. The concavities of the foot are obliterated first, the bony prominences later. Markedly swollen feet are often said to be club-shaped. The heel and the toes usually remain distinct. The distance between the toes may

be increased or the relative directions of the toes altered, and according to the amount of swelling of the sole they may be elevated from the floor when the foot rests on it. Talipes equinus may be present. In advanced cases the foot may appear as a globose mass of two or three times normal size.

The presence of fistulas through which a peculiar fluid containing grains escapes is characteristic of the mycetomas. The fistulas become manifest on spontaneous ulceration of the skin with resulting rupture of the abscesses. This usually takes place at the apex of a nodule. Fluid containing the grains continues to drain for a few days and then ceases, allowing the mouths of the fistulas to crust over and heal with the formation of scars. The fluid is seropurulent, sanguinolent, often sanious, and contains small grains. By probing, the fistulas are found to originate from deep small abscesses and to pursue tortuous courses to the surface.

Subjective symptoms are few and slight. Pain is rarely present even on manipulation of the affected part, on pressure sufficient to express the grains, or on probing the fistulas. Local tenderness often precedes rupture and drainage of the fistulas, and in some cases sensations of fulness, numbness, heaviness or stiffness are present. The normal sensation in the skin between the lesions is unaltered. In cases with marked swelling or deformity there may be interference with locomotion.

General systemic reactions, including fever, are usually absent. Adenopathy is uncommon. In some advanced cases, there is wasting of the muscles of the leg and rarely cachexia. The systemic reaction, when present, is probably the result of secondary infection.

Considering the multiplicity of causative organisms, the clinical types of mycetomas are strikingly few. As Bocarro's analysis revealed, in most cases the disease is limited to the extremities and in the greater portion of these to the foot. In his series of 100 cases, the foot was affected in 93 instances, the hand in 3, the leg in 2 and the trunk in 2. The color of the grains is one of the most striking features of any given case, and formerly was employed erroneously as the basis for etiologic classification. The species *Actinomyces bovis* is the cause of the disease commonly affecting the facial and cervical regions of man for which the term actinomycosis is usually employed. The species also affects the walls of the thorax and abdomen, the thoracic and abdominal viscera, and rarely the brain. In cattle it produces the well known condition of lumpy jaw or wooden tongue. Regardless of the variety of anatomic locations of these diseases, the clinical features are rather uniform.

In a typical case of mycetoma the patient may be aware of slight local tenderness at the site of a previous injury for about two weeks before the appearance of swelling. The swelling appears first as a small nodule and gradually extends peripherally as other nodules appear.

The older nodules in succession become soft at their apexes, and after a few days spontaneous rupture of the skin takes place with escape of the peculiar fluid containing the characteristic grains. The quantity of the fluid is at first abundant but gradually diminishes after a few days until the fistula crusts over and heals. A small scar marks the site of the fistula. As other nodules undergo the same evolution all stages are usually represented. With gradual extension of the disease, swelling increases and globose deformity of the part ensues. Inconvenience in locomotion results from the size or situation of the tumor or from destruction of the skeletal structures. Cases of twenty-five years' standing have been reported.

DIAGNOSIS

The direct diagnosis of mycetoma is simple, provided the possibility of its existence is considered. The swelling, fistulas and grains are often noted by the patient himself, or are easily demonstrable on pressure applied to the part or on probing of a fistula. The fungous nature of the disease is readily apparent on microscopic examination of the grains. Teased specimens for such examination are quickly prepared from the soft grains; the hard may be softened by soaking or boiling in caustic potash, or soda, sodium hypobromite, freshly prepared antiformin or eau de Javelle. In such preparations the mycelia are readily seen.

The mycetomas are most likely to be confused in America with tuberculosis, or malignant disease, and in tropical zones also with elephantiasis and yaws. Tuberculosis should be excludable on the absence of fever or by inoculation of a guinea-pig. Sarcoma and other malignant tumors seldom present multiple or transitory ulcers, and biopsy usually may be relied on. In cases of elephantiasis the swelling involves the leg, and there is neither ulceration nor discharge. In yaws there is a general reaction, including fever and a secondary eruption.

The distinction between the actinomycoses and the maduromycoses is made on the appearance of the hyphae. In the maduromycoses, the hyphae are large and segmented, and they possess well defined walls and usually chlamydospores. In the actinomycoses, they are fine and non-segmented, with neither well defined walls nor chlamydospores.

PROGNOSIS

The prognosis for cure depends on complete removal of the diseased tissues, as the course is chronic and the mycetomas do not tend to spontaneous cure. After complete removal by excision or amputation, recovery is rapid and there is no tendency toward recurrence in remote regions. The mycetomas are seldom the cause of death except by cachexia or secondary infection.

TREATMENT

The treatment of the mycetomas is surgical. Woolrabe [19] and Audrain [20] have ascribed cures to antiseptics (iodine and mercurochrome-220 soluble, respectively), but their observations are inadequate Other antiseptics and germicides and various internal medications, including potassium iodide, copper sulphate, bismuth sodium tartrate, copper citrate [21] and arsphenamine, have been tried without positive beneficial results. Thymol and oil of cinnamon, as applied to other fungous conditions, have been suggested by Gammel. Cauterization has not proved satisfactory.

In early cases, local excision may be sufficient and should be tried, but in advanced cases amputation is the only known curative measure

19. Woolrabe, Frederick: Curability of Madura Foot, J. Trop. Med. & Hyg. **21**:146, 1918.

20. Audrain, L. C.: A Case of Mycetoma, J. A. M. A. **83**:1165 (Oct. 11) 1924.

21. Palmer, F. J.: A Case of Madura Foot Treated by Chemotherapy: Apparent Cure, Indian Med. Gaz. **61**:74, 1926.

A REVIEW OF UROLOGIC SURGERY

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KIDNEY

Anomalies.—Surraco [1] reported a case of crossed ectopic kidney The right kidney was normal and in good position. The left kidney, which is the one usually displaced, was crossed and partially fused with the lower pole of the right. Both kidneys had lost their normal shape; the pelves were on the anterior surface and the ureteropelvic junctures were lateral to the kidneys. The ureter to the left kidney crossed the median line at the level of the second sacral vertebra; this is important embryologically as it suggests the area in which the ureteral bud found its first obstacle in its ascending progression.

[ED. NOTE.—Ectopic kidneys of this type do not usually cause symptoms, but at times, owing to their bulk and situation, there is considerable pain. Caulk [2] reported a case in which suspension and fixation of the kidney to the lumbar fascia partly relieved pelvic pain and a constant desire to urinate.

Stewart and Lodge [3] reviewed twenty-eight cases of unilateral fused kidney. They found that the displaced organ generally lies below the normally situated one, its upper pole fused with the lower pole of the latter. The ureter from the upper kidney generally enters the same side of the bladder. Usually three or four arteries pass to the fused organ, of which two supply the upper and two the lower half. One or both vessels to the upper part of the kidney arise directly from the aorta, in or below the normal position. The vessels for the lower part of the

1. Surraco, L. A.: Considérations sur le rein ectopique croise, J. d'urol. méd. et chir. **23**:411, 1927.

2. Caulk, J. R.: Surgery of the Ectopic Kidney, Ann. Surg. **78**:65, 1923.

3. Stewart, M. J., and Lodge, S. D.: Unilateral Fused Kidney and Allied Renal Malformations, Brit. J. Surg. **11**:27, 1923.

kidney were more irregular. They usually arose from the lower part of the aorta, at the level of the bifurcation, or from the common iliac of the opposite side.]

Kuksinskaja [4] stated that supernumerary kidneys are the most rare of the renal anomalies. A case is reported in which the supernumerary kidney was also cystic. A survey of the literature revealed twelve similar cases.

The supernumerary kidney is a definite, individual organ, usually found beneath the normally placed kidney. It has its own pelvis, ureter and blood vessels. The ureter may combine with the other ureter and open either into the bladder or into the external genitalia of women or the posterior urethra of men. Usually there are no symptoms to indicate their presence. If infected, the usual clinical symptoms, such as pain, swelling, hematuria or dysuria, are present. The surest method of diagnosis is by cystoscopy and pyelography. Kuksinskaja reported a case of a woman, aged 43; she complained of pain in the right flank radiating into the ileocecal region. Examination and cystoscopy led to the diagnosis of cyst or tumor of a ptotic right kidney. Operation showed a supernumerary cyst of the kidney, the normal right kidney was also found. The cystic kidney, which was 10 by 13 by 5 cm., contained 200 cc. of fluid; the cyst was about 9 cm. in diameter on the convex surface of the kidney. Histologically, it was lined with a flat type of epithelium. Proliferative activity was not noted, and a definite cause for the cyst could not be found.

Lazarus [5] reported a case of unilateral fused kidney in which heminephrectomy for calculus pyonephrosis was performed. The diagnosis was made by pyelographic studies. At operation, the renal mass was found to extend from under the arch of the diaphragm to just below the crest of the ilium. When it was freed from its perinephritic coverings, it was seen to be made up of two parts. The upper portion, which comprised a little more than two thirds of the entire renal volume, was solid and appeared to be normal renal structure. The lower portion consisted of a fluctuating cystic sac and was densely adherent to the perirenal tissues (perinephritis). It was freed only with the greatest difficulty. There was a well defined line of demarcation evidenced by a superficial groove crossing the mass on its anterior surface, which indicated the site of fusion of the two kidneys. The upper kidney presented a small extrarenal pelvis and a normal appear-

4. Kuksinskaja, G. F.: Cyste einer überzähligen Niere, Ztschr. f. Urol. **21**: 342, 1927.

5. Lazarus, J. A.: Unilateral Fused Kidney, with Report of a Case in Which Heminephrectomy for Calculus Pyonephronis was Performed, J. Urol. **18**:269, 1927.

ing ureter which passed over the anterior surface of the mass. The pelvis belonging to the ectopic portion was greatly dilated and wrapped up in a mass of inflammatory fat. Its ureter was of normal size and was seen lying on the anterior surface of the kidney and proceeding downward and to the left. The pelvis was dissected free of adhesions with great difficulty and its blood supply inspected. There were two sets of large vessels entering the fused kidney. The upper set, arising from the aorta and vena cava, crossed the upper border of the dilated pelvis of the ectopic kidney and entered the hilum of the right kidney. The lower set arose from the right common iliac and coursed along the lower border of the dilated pelvis to enter the kidney in its lateral surface. These vessels were clamped, cut, doubly ligated and divided. The ectopic kidney was freed with great difficulty and delivered, and during this procedure several aberrant vessels were encountered. These were ligated and divided and the ectopic kidney resected through the groove indicating the line of fusion. The divided end of the upper kidney was closed with three mattress sutures of plain catgut. The kidney was then decapsulated, and through a small nephrotomy wound a tube was introduced into the renal pelvis. The kidney was finally replaced in its bed and carefully surrounded with perirenal fat. The patient left the hospital with the wound practically healed.

Tumors.—Wollstein [6] reported a series of eighteen renal tumors in children, their ages varying from 3¾ months to 6 years; seven were in the first year; two were more than 3 years of age, and nine were between 1 and 2. Ten were girls and eight were boys. Ten of the tumors were in the right kidney and eight were in the left. All tumors were unilateral, retroperitoneal and within the renal capsule.

Wollstein noted that these tumors may be in any portion of the kidney. Microscopically, all were embryonic; one was an unmixed spindle cell sarcoma, thirteen were adenosarcomas, three were leiomyo-adenosarcomas, and one was a rhabdomyo-adenosarcoma. Embryonal renal neoplasms occurring in young children are a heterogeneous group, being closely related but not identical histologically or histogenetically. The fully developed renal tissue does not have any part in the formation of tumor. The author adds that the more solid tumors are more easily removed because they remain within their capsules. Four children who survived operation ten months or more had this type of tumor. Metastasis into the lungs and liver may develop into the firm type of growth as well as the softer ones, but metastasis appears less frequently and less early in the former type. One patient survived to adult life, and one is well six years after operation. The growth in the former was

6. Wollstein, Martha: Renal Neoplasms in Young Children, Arch. Path. **3**:1 (Jan.) 1927.

a rhabdomyo-adenosarcoma, in the latter adenosarcoma, showing more adenomatous than sarcomatous differentiation. The prognosis in cases of primary renal neoplasms in young children is therefore not entirely hopeless.

[ED. NOTE.—It is well known that the kidney is not an infrequent site for malignant disease during infancy and childhood. The tumor found in most cases is of the mixed embryonic type, although many and varied names have been applied. In many respects these tumors have and still present the problems and diversities of malignant tumors of the testicle. This is not at all surprising when the fact is considered that during the embryologic development of the organism, the genital and urinary tracts originate in close proximity, namely, in the urogenital fold in the anlage of both the mesonephros and the genital gland. These tumors, mixed in constituent tissues, as with testicular tumors, are highly malignant and great destroyers of life. They are a singular and characteristic growth and have presented many problems in pathogenesis.

The relative occurrence of renal tumor in children is 0.06 per cent (one in 1,600); in adults it is 0.25 per cent (one in 400). The relative frequency of renal tumors among tumors in general in children is 20.4 per cent (one in five); in adults from 0.5 to 2 per cent. Involvement as to the side and sex is about equal, 4 per cent being bilateral. The usual growths are "embryonic mixed tumors," most of them occurring within the first five years of life, although they have been found at birth and in later life.

Three hypotheses have been advanced as to pathogenesis. 1. The tumors are due to inclusions of the wolffian body (Birch-Hirschfeld). 2. They are aberrant cells from the myotome, sclerotome and mesenchyme (Wilms). 3. They are descendant from the true embryonic renal tissue or renal blastema and develop by a process of metaplasia (Busse, Muus, Ewing). These "embryonic mixed tumors" may assume large proportions, the average being from 1 to 4 pounds (0.5 to 1.8 Kg.) The pathologic picture presented is an embryonic one, dominated by either glandular or cellular tissue; if the former, it has been called embryonal adenocarcinoma; if the latter, embryonal adenosarcoma.

The clinical picture is presented usually in the following order: abdominal tumor; presence or absence of pain or hematuria, anemia, general weakness, anorexia and cachexia terminally; and either local or distant metastasis. Tumor nearly always is the initial sign. It is distinguished from neurocytoma, retroperitoneal tumor, enlargement of the spleen, tumor of the liver, hydronephrosis and ovarian lesions. The prognosis is poor, 95 per cent of the patients ultimately dying. The treatment is early diagnosis and radical excision. The use of the roentgen ray has given little encouragement.]

Nicholson [7] noted that renal or abdominal symptoms accompanied by fever, are not necessarily indicative of new growth. There is much evidence, however, to show that fever is associated with uncomplicated neoplasms, although not as frequently as with inflammatory disease.

In a review of the literature it was found that Briggs noted fever in 38 per cent of cases of new growth and in 45 per cent of cases in which ulceration had occurred; renal malignancy, however, was not mentioned. Israël, in 146 cases of tumor of the kidney and suprarenal glands noted fever in 8 per cent of the uncomplicated cases. Israël felt that the cause of the fever must be looked for either in the rapid growth of the tumor cells or in the destruction of normal tissue. Necrobiosis, which occurs in all malignant tumors, should not always be considered the cause of the fever since fever is not always present even when this occurs. Voelcker stated that fever is not infrequently present with hypernephroma. There were cases extending over a period of a week with temperatures as high as 102 F. Cases were noted in which the temperature was the initial sign of the disease. In speculating as to the cause, Voelcker refers to the absorption of albumin bodies produced by the process of involution, also mentioning as a possibility that inflammation of the renal pelvis plays a part, because this structure is often invaded by plugs of necrotic new growth. Berg believed that the great size and rapid growth of hypernephroma were factors in the cause of the increased temperature. Castano, in a series of seven cases, found that chills had occurred in two and irregular fever in one. In each case, fever subsided following nephrectomy.

Nicholson observed a case, that of a woman, aged 38, who had had fever almost continuously, intermittent abdominal pain and general weakness. A correct diagnosis had not been made in spite of careful observation over a period of eleven months. Roentgen-ray examination then showed widening of the hilum of the lung. Seven months later, eighteen months after the onset of the illness, annular opaque areas appeared in the lung and a diagnosis of tumor, probably Hodgkin's disease, was made. Fever had been present almost continuously for twenty months. Necropsy showed carcinoma of the lower pole of the right kidney with metastasis to the left suprarenal gland, spleen, liver and lungs.

Cullen [8] reported a case of fibrolipoma simulating tumor of the right kidney. The patient was a small girl having signs of digestive disturbance and pain in the right lower quadrant. There were definite

7. Nicholson, Daniel: Fever with Renal Carcinoma, Arch. Path. **3**:393 (March) 1927.

8. Cullen, T. S.: A Fibrolipoma Closely Simulating in Form and Location a Tumor of the Right Kidney; Subacute Appendicitis, Surg. Gynec. Obst. **45**: 152, 1927.

signs of mild appendicitis, and a firm lobulated tumor was palpated in the right side of the abdomen. The tumor extended up under the rib and well down within the crest of the ilium. A diagnosis of tumor of the right kidney was made. The usual incision was made and the tumor, which was lobulated, was shelled out; the normal sized kidney, which was not connected with the tumor, was pushed up under the ribs.

The tumor, which was extraperitoneal, was removed and apparently consisted of hard lobulated fat. The appendix was removed through the same incision. The tumor was a typical fibrolipoma.

Tumors of the Renal Pelvis.—Thomson-Walker [9] reported a case of squamous cell carcinoma of the renal pelvis associated with renal calculus. The patient, a man, aged 63, had passed calculi at intervals for twenty-seven years. For two years stabbing lumbar pain had occurred and recently severe attacks of pain about once a fortnight. There had never been hematuria. The right kidney was much enlarged. There was increased frequency of micturition, and the urine contained a heavy deposit of pus and gave a pure culture of *Staphylococcus albus*. The roentgen ray showed a large, heavy, irregularly rounded shadow with a somewhat ill defined edge in the right lumbar area. Right nephrectomy was followed by uninterrupted recovery.

The kidney had the external appearance of a large calculous pyonephrosis. On incision, a quantity of pus and débris escaped from the dilated pelvis. A large, rounded calculus with a rough surface lay in the pelvis in a bed of grayish-white shaggy material resembling asbestos. This material lined the middle third of the pelvis and extended into the slightly dilated calices at this level. It was from 0.3 to 0.6 cm. thick. The surface was shaggy and the upper and lower edges were sharply defined. The renal pelvis and calices above the affected areas were dilated. Microscopic examination of the asbestos-like lining showed leukoplakia which passed into squamous cell carcinoma with cell nests. The infiltration had not penetrated beyond the muscular wall of the pelvis and there was no infiltration of the renal substance. Enlarged lymph nodes were not found at operation.

[ED. NOTE.—Squamous cell tumors of the renal pelvis probably result from chronic irritation. Not infrequently, as in this case, there is a history of the presence of renal stone for a long time. These tumors are highly malignant, rapidly involve the renal parenchyma and adjacent tissues, and metastasize readily. Owing to the lack of symptoms suggesting a malignant condition, these tumors are rarely seen when the growth is small or localized to the renal pelvis. In 108 cases of associated renal calculi and malignancy, reviewed by Martin and Mertz, the

9. Thomson-Walker, John: Squamous Carcinoma of the Renal Pelvis Associated with Renal Calculus, Proc. Roy. Soc. Med., Sect. Urol. **20**:20, 1927.

symptoms suggestive of stone averaged nineteen years in duration; the symptoms suggestive of malignancy usually had been present about five months. Most stones found in cases of carcinoma of the renal pelvis are rough and irregular; they are usually of the staghorn, earthy phosphate type, and may be extremely large. Kaufmann reported a case in which the stone weighed 93 Gm. A similar case was noted by Oraison, in which the patient had had attacks of renal colic for twenty-eight years; the stone weighed 107 Gm. Wells and Kundrat also reported cases of squamous cell tumors associated with large branching stones. Stones occurred in six of eleven cases in which the condition was considered by Wells as definite squamous cell carcinoma. The operative mortality is high, and the majority of patients who survive die shortly after the operation for local recurrence, or metastatic growth.

Five cases of squamous cell tumor of the renal pelvis were seen at the Mayo Clinic between 1907 and 1922. Renal stone was associated in four of the cases. The stones in three kidneys were extremely large and of the staghorn type. One patient died eight days after operation; three others died during the first four months. The fifth patient is alive and without symptoms of recurrence six months after operation.]

Hunt [10] stated that twenty-three of 318 malignant tumors of the kidney removed at the Mayo Clinic were primary in the renal pelvis. Eight of these were highly malignant sessile epitheliomas, the remainder were relatively benign papillary epitheliomas. All of the first group of patients (seen since 1910) have died. The results in the latter group have been better; six patients are living and well, three less than a year later, and three for seven, five and three years, respectively. Of the nine who died, one lived seven and a half years, one lived four and a half years, one three and a half years, one two and a half years and five less than one year, after operation. Sessile epithelioma progresses and extends by invasion into perirenal tissues and renal veins, and metastasizes remotely, while the papillary type progresses by extension along the mucous membrane of the calices, ureter and bladder.

Hematuria is the most constant symptom of papillary tumor. It occurred in all of the cases in this series. The presence of a palpable enlarged kidney is usually dependent on the presence of hydronephrosis. A large renal tumor was palpable in five of the cases, and in each instance was due to hydronephrosis. The presence of a papillary tumor of the bladder, at or near the ureteral orifice, as seen on cystoscopic examination, should immediately give the clue to the diagnosis. Such a tumor was present in but four cases on primary cystoscopic examination; in a fifth case a small amount of papillary tissue projected from the

10. Hunt, V. C.: Papillary Epithelioma of the Renal Pelvis, J. Urol. **18**: 225, 1927.

ureteral orifice. In cases in which transplantation to the bladder has not occurred, the diagnosis of renal tumor may usually be established from the pyelogram.

Four patients in the series were treated by simple nephrectomy. One of these is living and well three years after operation. One patient died after four years with extensive recurrence in the bladder; two patients died at the end of five and six months, respectively. One patient is living and well seven years after nephrectomy and partial ureterectomy. There were eight cases in which complete nephro-ureterectomy was performed, the ureterectomy from two weeks to as late as twenty-one months after nephrectomy. In four of these, it was necessary to perform simultaneous segmental resection of the bladder for vesical metastasis that had occurred since the nephrectomy. In this series two patients died in the hospital, one from pulmonary embolism on the eleventh day, and the other on the thirteenth day from pelvic peritonitis resulting from a necessary transperitoneal resection of the bladder. One patient of this group was living and free from recurrence at a recent examination five and a half years after operation; another is living four months after operation. Three patients who died from recurrence lived two and a half, three and a half, and seven and a half years, respectively, following late ureterectomy and resection of the bladder for metastasis.

On account of the frequency and involvement of the ureterovesical segment, Hunt believed that segmental resection of the portion of the bladder to include the intramural portion of the ureter and the adjacent area simultaneously with nephro-ureterectomy seems necessary to insure the best prognosis. The results seem to show that the interval between nephrectomy and subsequent ureterectomy is an important factor in the prognosis, and is readily obviated by one stage complete operation of nephro-ureterectomy with segmental resection of the bladder.

Hunt stated that if the diagnosis of papillary epithelioma of the renal pelvis has been accurately established by the presence of metastasis to the bladder, it is desirable to proceed with exposure of the lower portion of the ureter and resection of the bladder before the kidney is removed. This facilitates the removal of the entire specimen intact. Through a low median-line incision extending from the pubes to the umbilicus extraperitoneal access to the lower two thirds of the ureter and bladder is readily afforded. The ureter is entirely freed, down to the ureterovesical juncture, and the base of the bladder is elevated to allow excision of the intramural portion of the ureter and an area of bladder wall around the growth. The resected segment of bladder and the lower portion of the ureter are wrapped in gauze and tucked high in the extraperitoneal space toward the kidney. After the bladder is

reconstructed and the anterior incision is closed, the patient is turned on the side, and through the posterolateral incision, which gives access to the upper third of the ureter, the kidney, ureter and bladder are removed intact.

Tumors of Renal Capsule.—Johansson[11] reported two cases of tumor of the renal capsule; one a fibroleiomyoma in a man, aged 57, the other a fibrolipoma in a woman, aged 47.

Pararenal tumors are relatively rare. About one half of all retroperitoneal lipomas originate from the tissues around the kidney, and only a small proportion of these start in the renal capsule. These tumors, often reaching considerable size, may grow in either a lateral or a medial direction, in the latter case extending widely into the mesentery, where there is least resistance. They may have little, if any, attachment to the kidney or else grow around it, encroaching on all sides.

In the absence of contraindications and if technically practicable, the treatment in such cases should be operative; if operation is not performed, one must reckon either with sarcomatous degeneration or else with sequences from the mere size of the tumor which alone may demand its removal. There are thus a number of cases described in which extirpation had been impossible because of technical reasons and in which the patients died later on account of mechanical disturbances created by the tumor, such as compression of the lungs, intestines or ureters, or from cachexia. At postmortem examination, one may also find that such a tumor was the cause of death.

The prognosis, however, even if the radical operation is performed, is far from favorable. The primary mortality is already high, often owing to technical difficulties with risk of hemorrhage and injuries to intestines. In a series of cases reviewed by Wahlendorf, death occurred in about 30 per cent, either as a direct result of operation or from some cause closely related to it. To this may be added the fairly great risk of sarcomatous degeneration. The operative cases have not been under observation sufficiently long to determine how great this risk may be.

Histologically, benign tumors also recur fairly frequently, and in a number renewal of operative procedures has been indicated. In certain cases this is probably explained by the fact that tumors have grown diffusely at an early stage and thereby prevented radical removal.

These tumors are generally of considerable size at the time of operation, and extraperitoneal exposure by a lumbar incision would therefore meet with fairly great difficulties. In Johansson's cases the tumors were exposed successfully by the transperitoneal route. On the

11. Johansson, Sven: Two Cases of Tumor of the Renal Capsule, Acta chir. Scandinav. **61**:197, 1926.

other hand, extraperitoneal incision over the abdomen, for example, the pararectal as suggested by Hoffmann for this purpose, should also be applicable.

[ED. NOTE.—Tumors of the renal capsule are not common. As a rule they are not described as such, but are grouped with other retroperitoneal tumors and cysts in the region of the kidney under the head of perirenal tumors. As regards the etiologic and pathologic factors of renal tumors in general, there is great divergence of opinion with regard to the origin of capsular tumors.

Reports of tumors of the renal capsule have rarely appeared in the American literature, the most recent report being by Day.[12] They can usually be classified in three groups: (1) connective tissue tumors of the renal capsule, (2) fatty tumors of the perirenal fat layer and (3) mixed malignant tumors in which occasionally sarcomatous elements predominate. Large fibromas occasionally grow from the capsule or the hilum of the kidney, but they are generally associated with fat, smooth muscle and tissue of the kidney and exhibit myxomatous changes; hence such terms as fibrolipoma, myolipoma, fibromyolipoma, fibroleiomyoma, and myxoma fibromatosum are noted. Lipoma has been found to be most common. Liebermann,[13] analyzing 165 tumors, found the proportion as follows: lipoma, 46 per cent; fibrolipoma, 20 per cent; myolipoma, 10 per cent; fibromyolipoma, 10 per cent, and sarcoma, 14 per cent.

Etiologically, the tumors arise as follows: (1) renal capsule gives rise to the various fibromas and sarcomas in the renal capsule, (2) lipomas in the renal fatty capsule, (3) the embryonic mixed type in the wolffian body and (4) rare hypernephroma in suprarenal rests. It should also be remembered that tumors of the hilum of the kidney that occur only rarely are of a similar nature, especially the lipomatous. Other hypotheses by various observers are of interest. Birch-Hirschfeld[14] has classified the tumors as embryonic adenosarcomas or sarcomatous glandular swelling, believing them to originate from the wolffian body. Haslinger believed that they may be due to pressure on the renal artery or to bleeding in the renal capsule followed by organization. Ewing[15] believed that the growths must owe their origin to some developmental disturbance of the kidney, the nature of which is undetermined.

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12. Day, R. V.: Perirenal Tumors, J. A. M. A. **80**:840 (March 26) 1923.

13. Liebermann, quoted by Haslinger: Zur Kasuistik der Nierenkapseltumoren, Ztschr. f. urol. Chir. **20**:89, 1926.

14. Birch-Hirschfeld, quoted by Haslinger: Zur Kasuistik der Nierenkapseltumoren, Ztschr. f. urol. Chir. **20**:89, 1926.

15. Ewing, James: Neoplastic Diseases, Philadelphia, W. B. Saunders Company, ed. 2, 1922, p. 165.

In connection with the wolffian body it is of interest that these tumors occur much more frequently in women. Hinman, Gibson, and Kutzmann [16] have discussed the rôle of the wolffian body as an etiologic factor and found that more evidence is gradually being collected to support the view that a great variety of cystic and solid neoplasms of the retroperitoneal space in relation to the suprarenal, kidney, broad ligament, spermatic cord, epididymis and possibly the testis have their origin in remnants of the wolffian body. In the complex development of the urogenital tract abundant opportunity for maldevelopment undoubtedly exists, while the numerous fetal remnants left over in the process of development afford an attractive explanation. Mesonephric cysts have been noted much oftener in females; this has been explained as being due to the relatively greater amount of the remnants of the wolffian body in the female as compared to the male. The latter utilizes most of the primitive urinary excretory apparatus in the formation of the vas deferens and epididymis, whereas in the female the major part of the wolffian body remains vestigial.

Clinically, these tumors produce few, if any, symptoms. Except for the sarcomas, they grow slowly, tending to encompass the kidney without invasion of the renal capsule. As a result, urinary symptoms are rare. These tumors will continue to grow and fill the lumbar and renal space with pressure encroachment on the adjacent organs; hence such symptoms as dyspnea, edema and constipation occur. The renal mass has been known to increase so as to weigh from 30 to 60 pounds (14 to 27 Kg.). Gastro-intestinal symptoms, especially nausea and vomiting, are usually the most prominent. Barring the absence of urinary disturbances, the physical signs are similar to those of renal tumors. The tumors occur with greatest frequency in adults between the third and fifth decades.

Preoperative diagnosis is rarely made, due to the number of other confusing conditions. The treatment is complete radical excision. This is not always technically possible because of the danger of injury to the adjacent vital organs as well as the possibility of extensive infiltration. For this reason the operative mortality is high (50 per cent). There is a decided tendency to recurrence of the benign as well as the malignant tumors; thus the ultimate prognosis is bad.]

Renal Tuberculosis.—Fullerton [17] reviewed his series of 150 cases of tuberculosis of the kidney and bladder. He believed that primary tuberculosis of the bladder rarely exists and that the sequence of events in many cases is infection of the kidney, followed by secondary involvement of the prostate, vesicula and epididymis.

16. Hinman, Frank; Gibson, T. E., and Kutzmann, A. A.: Cysts of the Wolffian Body, Ann. Surg. **79**:762, 1924.

17. Fullerton, Andrew: Tuberculosis of the Bladder and Kidney, Irish J. M. Sc., 1927, no. 13, pp. 5-14.

One of the most striking phenomena in his cases of unilateral tuberculosis is the occurrence of what he terms "unilateral diuresis." This is the discharge from the affected side of urine of low specific gravity, often as low as 1.000 to 1.005, as compared with 1.015 to 1.025, or more, on the sound side. The difference in quantity, specific gravity and the reaction on the two sides is striking, and occurred, in his experience, in over 90 per cent of the cases. On the diseased side there is a free flow of pale urine, faintly acid, neutral or even alkaline in reaction, of low specific gravity, containing albumin and pus, while on the sound side there is a sluggish flow of highly acid urine of high specific gravity, often depositing urates on standing. There may be a small amount of albumin on the nontuberculous side, but, failing other evidence of renal inefficiency, he disregarded this in prognosis. If the diseased kidney is removed, the albumin disappears from the sound side, being apparently due to toxins and not to any serious impairment of the remaining organ.

In the rare cases in which cystoscopy and ureteral catheterization are impossible, Fullerton adopts the method of cutting down on the ureter in the iliac region, and catheterizing the ureter through a small slit, which is then closed immediately. This operation is performed by employing the muscle-splitting incision commonly used for removing the appendix. The peritoneum is not opened, but is peeled inward from the lateral wall of the abdomen toward the median line. The ureter remains attached to the peritoneum, and, when recognized, is seized, without being detached, by two special ring forceps by means of which it can be brought up into the wound. A small slit is made and a catheter is introduced. On removal of the catheter, the small aperture is carefully closed by two rows of fine catgut suture. If the ureter is found to be thickened, it is not further disturbed, as it is obviously the ureter of the affected kidney. All that is necessary is to demonstrate the presence and efficiency of a second kidney. Fullerton has performed this operation several times in tuberculous cases.

Fullerton believed that, from a clinical point of view, cure is possible in certain cases without operation. He reported several cases under observation for years in which operation was not performed. Improvement had occurred, although in most cases the symptoms demonstrated that the original lesion was still present. In the cases in which operation has been performed remissions occur, but the general tendency of symptoms is to grow worse.

Fullerton stated that present knowledge justifies the recommendation of nephrectomy for renal tuberculosis with or without secondary involvement of the bladder as being the best and surest method of cure. If done early, before the bladder is involved, the relief is often immediate, and in any case the patient has a better chance of cure and is less

susceptible to the crippling effects of scar tissue in the bladder; his immunity from recurrence of the growth elsewhere is also greater.

Ide [18] stated that renal tuberculosis, which usually remains unilateral for a long time, should be treated by nephrectomy. Edema and hypertension are positive signs of bilaterality and exclude surgical intervention.

Medlar stated that renal tuberculosis may heal spontaneously in the first stages, and that it is usually present in fatal cases of pulmonary tuberculosis. In twenty-two of thirty cases of fatal pulmonary infection, tuberculosis bacilli were found in the kidney, usually in the cortex. The kidney follows the same laws of tuberculization as the lungs which in the majority of cases do not undergo caseation.

Stevens [19] reported a case of renal tuberculosis in which the inoculation tests on guinea-pigs were positive with urine taken from both the right and the left kidney. Following right nephrectomy, repeated inoculation tests with urine from the remaining organ were negative, and when last seen, one and a half years later, the patient was apparently in good health.

Apparently normal kidneys may sometimes permit the bacilli of tuberculosis to pass. Hobbs reported that they were found in the urine of more than 8 per cent of 422 patients suffering from pulmonary tuberculosis, but without any other symptoms referable to the urinary tract. The kidney may be definitely involved without causing symptoms. In 143 cases of advanced pulmonary tuberculosis, involvement of the kidneys could be detected microscopically in 55 per cent; in only a few were symptoms of renal tuberculosis present during life. In 3,766 other cases of pulmonary tuberculosis reported by various observers, renal involvement was noted microscopically at necropsy in 588, or in a little more than 15.5 per cent. Wildbolz, stressing the value of functional renal tests, stated that in more than 500 nephrectomies he had not been misled in a single instance by the use of indigo carmine or cryoscopy as functional tests, and that the questions whether or not renal tuberculosis is present and, if so, whether unilateral or bilateral, are to be answered with certainty only by means of functional tests of the kidneys. He does not consider a small amount of pus and a few bacilli of tuberculosis pathognomonic of renal tuberculosis unless there is a decrease in function.

Stevens concluded that repeated examination of the urine by means of smears and inoculation of guinea-pigs are sometimes necessary in order to discover the bacilli of tuberculosis. Roentgenograms are positive in a much larger number of cases than is generally appreciated, and they should be more frequently employed in the diagnosis of renal tuberculosis.

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18. Ide: Tuberculose renale, Rev. méd. de Louvain, 1927, no. 3, pp. 47-49.

19. Stevens, W. E.: Diagnosis of Renal Tuberculosis, J. A. M. A. **88:71** (Jan. 8) 1927.

Tuberculosis of the kidneys is more common in children than would appear from the literature, and modern urologic diagnostic procedures, such as cystoscopy, ureteral catheterization, tests of renal function and pyelo-ureterography, are frequently indicated in infants and children as well as in adults.

Lazarus [20] reported a case of tuberculous horseshoe kidney. At operation the left segment of the kidney was found to be moderately enlarged and close to the lumbar spine. The upper pole was adherent to the under surface of the diaphragm. The ureter was greatly thickened and on section the typical cheesy necrosis characteristic of tuberculosis was noted. An attempt to expose the lower pole of the kidney was futile. The ureter was severed between ligatures and by tracing the lower part of the kidney it was found to take an inward course toward the median line. By carefully pushing the posterior parietal peritoneum mesially, one could distinctly see a thick isthmus of renal tissue crossing over the aorta and vena cava and merging with the kidney on the opposite side.

With the diagnosis of horseshoe kidney established, the pedicle and all the accessory vessels were doubly ligated and divided and the kidney gradually liberated from above downward. By careful digital dissection, the isthmus was separated from the aorta and vena cava and divided between two clamps. The clamps were then removed and a few mattress sutures introduced into the divided end of the isthmus. One tube was introduced into the renal fossa, and the wound was closed in layers.

The specimen, when opened, showed a large tuberculous abscess in the upper pole of the kidney filled with pus and cheesy necrotic material. The cortex over this cavity was much attenuated.

Roschdestwensky [21] (Urologic Clinic of the Tuberculosis Institute of Leningrad) found in the clinical and necropsy examination of 230 cases of tuberculosis of the genito-urinary tract that in all clinically examined cases pulmonary tuberculosis was more or less pronounced; that in the majority of cases the pulmonary process was stationary and well localized, and played only a minor part in the entire picture, and that in well defined cases, specific involvement of urinary and genital tracts was comparatively rare. If the urinary and genital tracts were definitely involved, pulmonary tuberculosis showed a favorable course; on the contrary, the genito-urinary tract was more immune against tuberculosis if pulmonary tuberculosis existed.

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20. Lazarus, J. A.: Tuberculosis in a Horseshoe Kidney, J. Urol. **18**:247, 1927.

21. Roschdestwensky, W. J.: Ueber den Einfluss der Tuberculose der Harn und Geschlechtsorgane auf den Verlauf der Linigentuberkulose, Beitr. z. Klin. d. Tuberk. **63**:112, 1926.

[ED. NOTE.—Braasch found evidence of tuberculosis in other parts of the body, either healed or active, in 244 of 346 cases of renal tuberculosis. He believes that it is questionable whether renal tuberculosis ever occurs unless there is tuberculosis in some other tissue. Coincident tuberculosis, particularly if healed or dormant, does not necessarily render the prognosis less favorable after nephrectomy; in fact, it may be regarded as evidence of the patient's good resistance. Pulmonary tuberculosis is one of the most common complications occurring with renal tuberculosis. Renal tuberculosis is not a common complication of active pulmonary tuberculosis; it occurred as a coincident infection in less than 5 per cent of such patients in the Mayo Clinic. Evidence of healed tuberculous lungs is present in fully a third of all patients afflicted with renal tuberculosis.

Runeberg,[22] in twenty-two years, has treated 126 patients who had tuberculosis by nephrectomy and eighty-seven without nephrectomy. In 55 per cent, the symptoms had existed for more than a year before tuberculosis was discovered. In 70 per cent, the onset was imperceptible; in 74 per cent, the onset was disturbance of urination; in 16.3 per cent, pain in the loin; in 5.8 per cent, hematuria. In two cases the first sign noticed was pyuria, and in three cases, malnutrition. In 33 per cent of the cases, the bladder was normal; in 96 per cent, the urine was acid. Albuminuria and pyuria were continually present. The irregular structure of the pus cells made the differential diagnosis simple. In fifteen cases, bacteria were not found in acid urine, which is not characteristic of tuberculosis. Many cases showed tuberculosis bacilli coming through the normal kidney; this ceased following nephrectomy. One patient did not wish to be operated on and became well following autonephrectomy. In all other cases in which operation was not performed, the process continued to a fatal termination.

Actinomycosis.—Bedrna and Pavlica[23] reported actinomycosis in a man, aged 43; examination had revealed pain in the left hip and a palpable tumor in the left side of the abdomen. There was diminished function of the left kidney and albuminuria. Roentgenograms and the afebrile course indicated a tumor, although spasticity of the ileopsoas muscle indicated infection. Objective or subjective symptoms of disease of the liver were not present. The kidney was removed, and a large active actinomycotic abscess was found. There were also many abscesses in the fatty capsule and the ileopsoas muscle. The patient died the third day after operation with signs of hepatic insufficiency. At necropsy two large abscesses were found in the liver. The primary

22. Runeberg, B.: Tuberkulose der Harnorgane, Finska Läkaresällsk. Handl. Helsingfors, 1926; abstr. Ztschr. f. Urol. **21**:380, 1927.

23. Bedrna, J., and Pavlica, F.: Aktinomykose der Niere, Časopis lékařů českých, 1926, no. 35-36; abstr. Ztschr. f. Urol. **21**:396, 1927.

focus was not found, but was believed to be in the gastro-intestinal tract. The position of the renal abscesses and the pyelographic picture bear out Israël's dictum that chronic actinomycotic abscesses of the kidney originate in the parenchyma as "excretionmycosis."

Bedrna and Pavlica reviewed twelve cases of large actinomycotic abscesses described in the literature as "primary kidney actinomycosis." These abscesses occur in two forms: as a tumor with perinephritic symptoms and sterile or cloudy urine and lymphatic involvement and as chronic pyelonephritis with lymphatic involvement and normal or slightly enlarged kidneys; pyelography usually suggests a tumor with this form. The cystoscopic picture may be that of chronic cystitis. The function of the affected kidney is diminished. The prostate is seldom affected.

The treatment consists in nephrectomy. From the reported case it is concluded that the liver must be carefully examined preoperatively. Other treatments mentioned are roentgenotherapy, administration of potassium iodide and copper sulphate and vaccination.

Renal Stone.—Simon [24] asserted that the formation of stone in the kidney following fracture of the spine and injury to the spinal cord is also accompanied by some type of renal disease. This may be the result either of primary injury to the kidney at the time the spine was fractured, of secondary injury through infection of the urinary tract or of ascending cystopyelitis, the result of injury to the bladder.

The type of stone which forms depends on the secretion of the urine from the injured kidney. There may, therefore, be several types. True urinary stone may form independently of injury to the spine by the deposition of urinary salts, and thereby blood clots or traumatically formed connective tissue in the renal parenchyma give rise to the nucleus; this would cause stones to be formed according to the colloid precipitation theory. The particular type of stone formation (oxalate or urate) depends on the crystalline body content of the urine at any one time. Spinal lesions have little influence on this.

Phosphate stones form secondarily to the decomposition and ammoniacal fermentation of urinary salts. These may be the result of lesions of the spinal cord resulting in paralysis of the bladder, ureter and pelvis, as well as in infection in the urinary tract. Their origination is perhaps favored by lesions of the spinal cord, while in such lesions there is an increased output of calcium phosphate.

Calcium stones form following fracture of the spine and injuries of the spinal cord, because of the absorption of the calcium salts from the fractured surfaces of the vertebrae and from the long bones of the extremities.

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24. Simon, E.: Ueber Kalksteinbildungen in der Niere nach Wirbel-Säulenbruch und Rückenmarkslämhung, Ztschr. f. Urol. **21**:444, 1927.

Renal Circulation.—Johansson [25] reported a case in which the gradual obliteration of the vessels of the hilum caused the formation of capsular collaterals. The case was one of chronic pyelonephritis with calculus. Nephrectomy was performed, and when the kidney was detached from inside the capsule it was found to lie free in the operator's hand without a trace of bleeding. The kidney was lying inside its own capsule as a kind of sequestrum even though its excretory function had been maintained. Pulsation was not felt in the vessels of the hilum. The pedicle was tied and the fibrotic capsule removed. The arteries of the hilum showed marked endarteritis with thickened intima, poor in cells but without obliteration. The veins were wide; one or two smaller branches were filled with recent thrombi. The arterial branches near the cortical limit showed increasing endarteritis, in most places almost obliterated and in some places completely obliterated.

Endarteritis was not marked in the capsular arteries, which were numerous and relatively large. Transitional branches of these vessels were observed to pass across toward the parenchyma of the kidney and in several places were traced for a distance of 2 mm. into the cortex

The partial obliteration of the vessels of the hilum caused hyaline degeneration of many glomeruli. The capsular vessels, which did not show any obliterative or endarteritic changes, had developed in great numbers not only in the capsule itself but in the superficial layers of the cortex with communicating branches of arteries and veins passing across.

Meyer and Singer [26] considered spontaneous perirenal hematoma as a definite entity usually necessitating immediate surgical intervention. This condition causes severe pain, signs of internal hemorrhage, formation of an enlarging tumor in the loin and, at times, hematuria and signs of peritoneal irritation.

The authors are disposed to regard perirenal hematoma as they do intestinal obstruction. The diagnosis of mechanical obstruction of the bowel being established, the indication for operation is usually clear. Preoperative knowledge of the etiology, although highly desirable, is not essential in the decision as to the form of management, whether medical or surgical, and when the presence of a perirenal hematoma is established, whether or not the cause is known, the case ordinarily becomes surgical.

Nephrectomy.—Patel [27] reported a case in which the vena cava was torn during the removal of a large tuberculous right kidney. An

25. Johansson, Sven: Question of Collateral Circulation in the Renal Capsule, Acta chir. Scandinav. **61**:181, 1926.

26. Meyer, K. A., and Singer, H. A.: Spontaneous Perirenal Haematoma and Aneurism of the Renal Artery, Surg. Gynec. Obst. **45**:300, 1927.

27. Patel: Ligature de la veine cave au cours d'une néphrectomie, Presse méd. **35**:633, 1927.

attempted lateral suture of the vena cava had to be abandoned as the patient was on the verge of collapse. The vena cava was then doubly ligated both above and below the tear. The superior ligature was just below the anastomosis of the left renal vein. The postoperative course was uneventful; the urine was not suppressed and not until the tenth day was there transient edema of the lower limbs.

Patel believed that the anterior route is desirable for nephrectomy; in this case, it permitted him more readily to carry out the necessary ligation. At this time, a case was discussed by Gayet in which the vena cava was torn during the removal of a large hydronephrotic sac. In this case also the tendency toward collapse made it necessary to abandon attempts to suture the vena cava. Gayet put a clamp on the tear and left it in place after closing the incision; the patient recovered without trouble.

Casper [28] reported two cases in which it was impossible to remove the kidney on account of extensive perirenal adhesions. The first case was that of a woman, aged 23, in whom pyonephrosis developed thirteen years after nephrotomy. The second case was one of infected hydronephrotic sac of long standing. The ureter was ligated and divided in both cases, the upper segment being sutured into the skin. Vesical symptoms cleared readily and there was no further trouble.

(To be continued)

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28. Casper, L.: (Fehler und Gefahren in der Chirurgie) Schwierige Nephrektomien, Zentralbl. f. Chir. **54**:79, 1927.

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